Study of ankle brachial index in systemic hypertension

A Dissertation Submitted to

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For the Award of the Degree of

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GOVERNMENT KILPAUK MEDICAL COLLEGE
CHENNAI
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BONAFIDE CERTIFICATE

This is to certify that **"Study of ankle brachial index in systemic hypertension"** is a bonafide work performed by **Dr.Deepa Avadhani**, Post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai-10, under my guidance and supervision in fulfilment of regulations of the Tamil Nadu Dr. M.G.R Medical university for the award of M.D. Degree Branch I (General Medicine).

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DECLARATION

I **Dr.DEEPA AVADHANI**, solemnly declare that the dissertation titled **"STUDY OF ANKLE BRACHIAL INDEX IN SYSTEMIC HYPERTENSION"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **Dr.S.Ushalakshmi M.D.**,**FMMC.,** Professor of Internal Medicine, Government Kilpauk Medical College and Hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine)**.

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INTRODUCTION
Hypertension is considered to be one of the leading causes of morbidity globally. Approximately 7.6 million deaths (13–15% of the total) and 92 million disability-adjusted life years globally are due to hypertension. Hypertension increases the risk of cardiovascular disease, renal failure, and peripheral arterial disease. It adds to the total burden of cardio vascular risk factors if other risk factors are also present.
Blood vessels are main structures contributing to the development of hypertension. At the same time they are the target organs for long standing elevated blood pressure. Hypertensive patients with peripheral arterial disease are found to have increased future cardiovascular events.
Hypertension holds the responsibility for most of the stroke deaths and coronary artery disease deaths in India. Hence early diagnosis and treatment of complications is very important so as to reduce the morbidity and mortality in the high risk population.
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ABSTRACT

Background

Asymptomatic peripheral artery disease is common in patients with systemic hypertension. Ankle brachial index is an easy method to detect asymptomatic peripheral artery disease. ABI of ≤ 0.9 indicates peripheral artery disease. Presence of peripheral artery disease indicates the presence of atherosclerosis and predicts high cardiovascular risk.

Aim: To study the effect of systemic hypertension on ankle brachial index and to assess the effect of age, sex, body mass index and duration of hypertension on ankle brachial index.

Methods: After excluding the patients with exclusion criteria 198 hypertensive patients who visited the department of medicine, Kilpauk medical college hospital were studied. Patient details were recorded as per the proforma which included age, sex, BMI, duration of hypertension, ankle brachial index. Data was maintained in master chart and analysed.

Results: Prevalence of asymptomatic PAD was 47% in this study. ABI was ≤0.9 in 46.8% of males in this study. ABI was ≤0.9 in 47.8% of females in this study. There was inverse correlation between age and ABI. There was inverse correlation between duration of hypertension and ABI. There was inverse correlation between pulse pressure and ABI. There was inverse correlation between ABI and BMI.

INTRODUCTION

Hypertension is considered to be one of the leading causes of morbidity globally. Approximately 7.6 million deaths (13–15% of the total) and 92 million disability-adjusted life years globally are due to hypertension. Hypertension increases the risk of cardiovascular disease, renal failure, and peripheral arterial disease. It adds to the total burden of cardio vascular risk factors if other risk factors are also present.

Blood vessels are main structures contributing to the development of hypertension. At the same time they are the target organs for long standing elevated blood pressure. Hypertensive patients with peripheral arterial disease are found to have increased future cardiovascular events.

Hypertension holds the responsibility for most of the stroke deaths and coronary artery disease deaths in India. Hence early diagnosis and treatment of complications is very important so as to reduce the morbidity and mortality in the high risk population.

Ankle brachial index (ABI) is a simple and non invasive tool to assess the peripheral artery disease. It indicates the presence of atherosclerosis in the peripheral blood vessel. It is a powerful indicator of generalized atherosclerotic disease including coronary arteries.

Diagnosing PAD is clinically important for these reasons:

The first is to identify patients with elevated risk of myocardial infarction or stroke irrespective of symptoms of PAD

The second is to treat the symptoms of PAD to reduce the functional disability and limb loss.

Peripheral artery disease usually presents with intermittent claudication. Peripheral artery disease becomes a marker for systemic vascular disease as atherosclerosis is the basic pathology in other vessels also. Thus it helps in predicting ongoing atherosclerosis in systemic vessels involving coronary, cerebral, and renal vessels which may lead to an elevated risk of events like myocardial infarction (MI), stroke, and death.

Ankle Brachial Index(ABI) helps in making reliable diagnosis of the peripheral artery disease. Ankle brachial index is a ratio having systolic blood pressure in the ankle as numerator and systolic blood pressure in the arm as denominator. Measurement of ABI is simple and requires a blood pressure apparatus and a hand held Doppler probe. An ABI of less <=0.9 is considered as low ABI .It indicates presence of peripheral artery disease.

Patients with peripheral artery disease (PAD) of the lower limbs are at higher risk of premature death related to cardiovascular events irrespective of presence of symptoms. Hence early detection of peripheral artery disease gains importance as these patients will benefit from cardiovascular risk factor modification.

Hypertension is a known risk factor for peripheral arterial disease.(PAD) along with diabetes and smoking. The present study aims at prevalence of this condition in hypertensive patients without co morbidities. The measurement of ABI is easy to take. It is non invasive and safe , thus patient friendly . Hence it can be used to screen for lower limb ischemia in hypertensive patients.

REVIEW OF LITERATURE

SYSTEMIC HYPERTENSION

Systemic hypertension

Hypertension is a non communicable disease. It is prevalent in both developing and developed countries. It is a chronic condition. It is of great concern as it is a major risk factor for coronary heart disease, stroke and other vascular complications.

Definitions

According to the Seventh Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7), the following are the definitions which can be used in the blood pressure evaluation:

- Systolic BP <120 and diastolic BP < 80 mm Hg is considered to be normal blood pressure.
- 120 to 139 mm Hg systolic or 80 to 89 mm Hg diastolic is considered as prehypertension
- *Hypertension*:

  Stage 1—140 to 159 mm Hg systolic or 90 to 99 mm Hg diastolic

  Stage 2— 160 mm Hg systolic or > 100 mm Hg diastolic

Epidemiologic studies have shown that there is a gradual but definite increase in cardiovascular risk with increasing blood pressure above even “normal” values of 110/75 mm Hg[1][4][5][6][7]. So the concept of prehypertension has emerged. These patients are at risk to develop overt hypertension, with those in the 130/80 to 139/89 mm Hg range being twice as likely to become hypertensive[1]. Patients who have isolated systolic hypertension will have systolic hypertension yet managed to have diastolic pressures lower than 90 mm Hg.

Hypertension due to an identifiable cause is known as secondary hypertension. Idiopathic or primary hypertension is referred to as essential hypertension. Approximately 25% of essential hypertension patients have low-renin essential hypertension.[8] It is found that such patients are more salt sensitive[9]. They are at relatively low risk for myocardial infarction[10] and less likely to respond to weight reduction[11]. They may be African American or elderly[12] ,and they may show a pronounced response to drugs like diuretics and calcium-channel blocker agents[13].

It is very important to detect secondary causes of hypertension when there is atypical presentation. Secondary hypertension should be considered when hypertension presents in a younger age or its not responding to usual antihypertensive medications. Different etiologies have to be considered and different tools for diagnosis should be judiciously employed as and when needed to arrive at the cause based on clinical suspicion.

The common causes of secondary hypertension are

1. renal disease
2. pheochromocytoma;
3. Cushing's syndrome
4. primary hyperaldosteronism
5. hypothyroidism
6. hyperthyroidism
7. oral contraceptives
8. hyperparathyroidism
9. coarctation of the aorta
10. obstructive sleep apnea syndrome .

If 24-hour average blood pressure is greater than 135/85 mm Hg or a daytime average of 140/90 mm Hg it is known as ambulatory hypertension. There are devices which measure the blood pressure throughout the day at definite predetermined intervals. These devices help in detecting ambulatory hypertension. These can be used to evaluate white coat hypertension.

Resistant hypertension is defined as a diastolic blood pressure greater than 95 to 100 mm Hg despite the usage of three or more antihypertensives. Malignant hypertension is a serious condition. It will have marked hypertension with retinal exudates, retinal hemorrhage or papilledema. It is associated with end organ damage.

SYSTEMIC HYPERTENSION AND CARDIOVASCULAR RISK

Systemic hypertension is a major risk for atherosclerosis[14]. There is clear evidence from epidemiologic studies that the cardiovascular risk increases gradually with the blood pressure greater than even "normal" values of 110/75 mm Hg[1][4][5][6][7]. It holds good more for individuals aged 40 to 70 years, with each increase of 20 mm Hg in systolic blood pressure or 10 mm Hg in diastolic blood pressure .

The risk of cardiovascular disease (CVD) increases by two fold with each increase of 20 mm Hg in systolic blood pressure or 10 mm Hg in diastolic blood pressure in individuals aged 50-70 years. Blood pressure lowering has been shown to improve the outcome.[16][17][18] . It is proved in clinical trials that stroke incidence is reduced by 35% to 40% with the help of anti hypertensive therapy.

Antihypertensive therapy helps reducing myocardial infarction incidence by 20% to 25% . Treating hypertension is shown to reduce heart failure incidence by more than 50%[19]. There is evidence that in patients with stage 1 hypertension with other cardio vascular disease risk factors , reducing systolic blood pressure 12 mm Hg for 10 years prevents one death for every 11 patients treated. If CVD or PAD or target end-

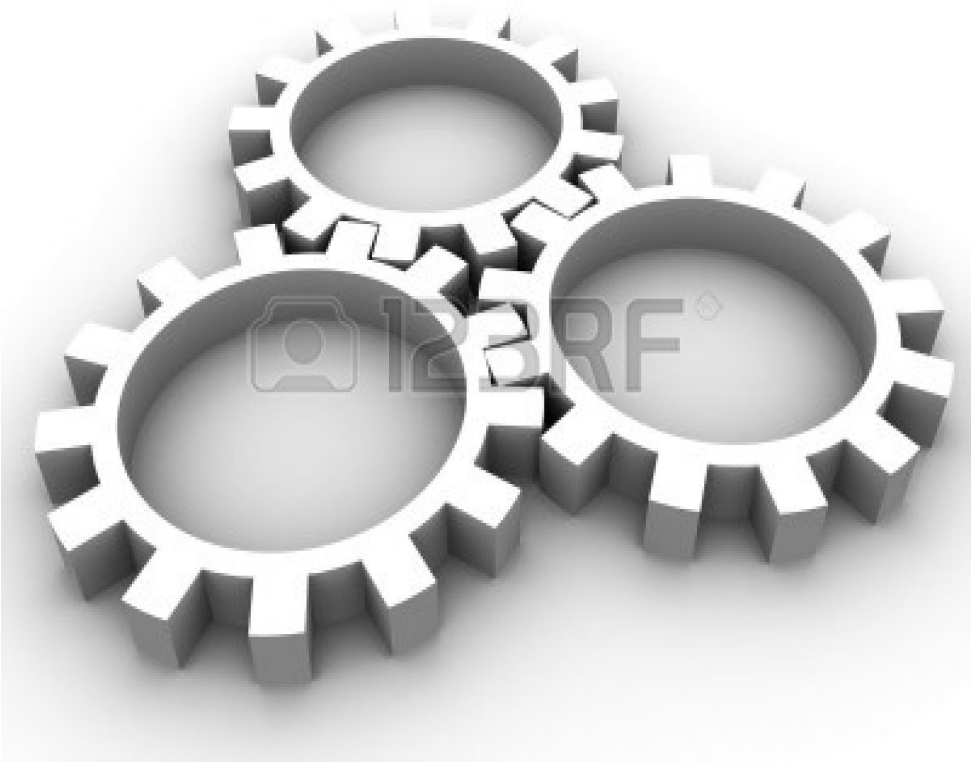
organ damage is present in these patients, to prevent one death 9 patients should be treated [20].

In patients with diabetes and chronic renal failure lowering the blood pressure is effective in prehypertensive stage[1]. Pulse pressure is also associated with increased risk[16][17][18]. As age advances arterial compliance decreases. This results in decrease in diastolic pressure and increase in systolic pressure. This is the reason why increased systolic and pulse pressure are more predictive of risk in older individuals[16] .Even with short-term therapy more beneficial effects of antihypertensive therapy can be witnessed in these subgroup of patients.[21][22] Systolic hypertension is the best predictor of cardiovascular risk in hypertensive patients aged 50 to 60 years, whereas it is the diastolic pressure in patients younger than 50 years[16].

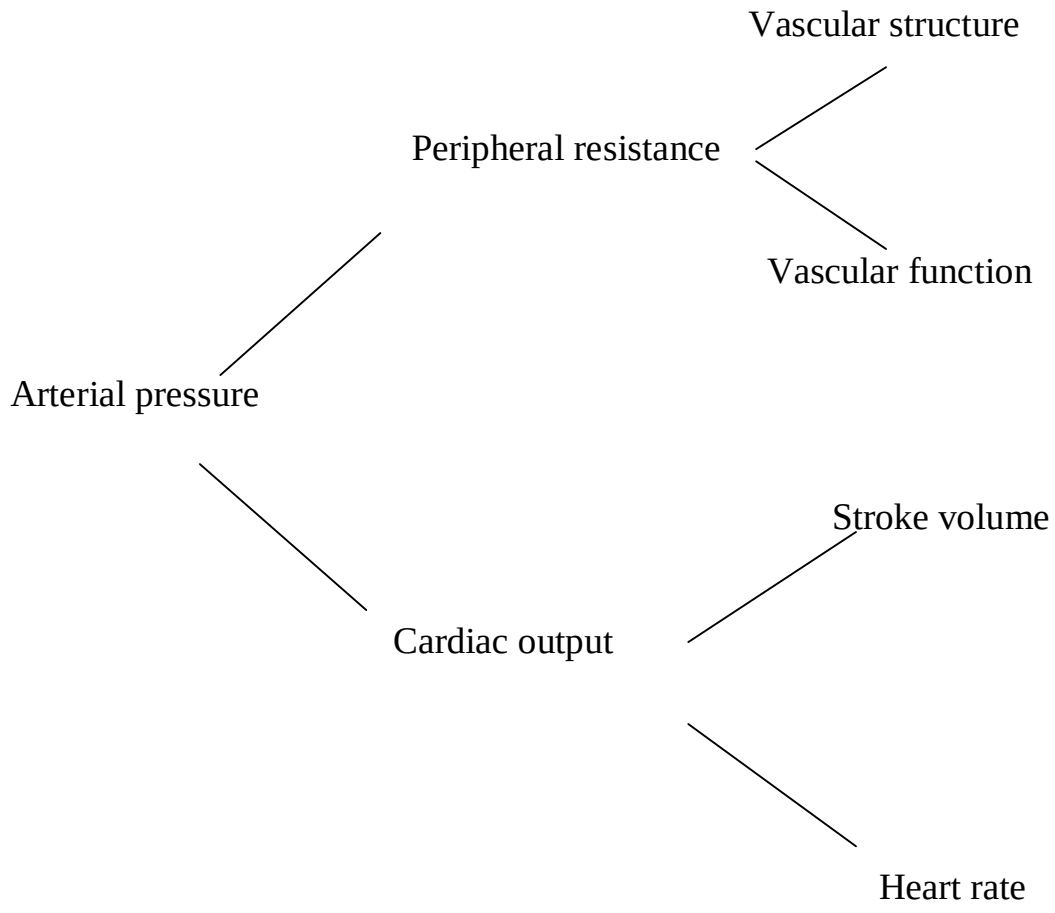
Control of diastolic blood pressures above 105 mm Hg is associated with reduced incidence of cardiovascular events (55% vs. 17% at 5 years)[23]. This benefit may not be as marked in patients with mild diastolic hypertension of 90 to 100 mm Hg. Hebert et al,[24]showed that treatment of mild diastolic hypertension for 4 to 5 years prevents a coronary event in 0.7% of subjects and a cerebro vascular event in 1.3%.This was a short duration study. In this cardiovascular death was lowered by only 0.8%. The benefit of treatment is better with long term

antihypertensive therapy. In fact, in Framingham Heart Study, the cardiovascular death risk for treated patients versus untreated patients was 0.4% over 10 years.[25]

MECHANISMS OF SYSTEMIC HYPERTENSION



It is important to know the mechanisms involved in regulation of normal blood pressure and elevated blood pressure to understand pathogenesis and treatment options. Both cardiac output and peripheral resistance are the two major factors involved in maintaining the arterial blood pressure .Stroke volume and heart rate are involved in determining the cardiac output. Contractility of the cardiac muscle and vascular compartment size contribute to the stroke volume.



Intravascular Volume

Vascular volume seems to be the major determinant of arterial blood pressure in the long run. Extracellular fluid volume is mainly dependant on sodium ion which is a major extracellular cation. When sodium chloride intake is more than the capacity of the kidneys to handle sodium, there will be initial expansion of intracellular volume and increase in cardiac output. Auto regulation of blood flow is seen in many vascular beds. This includes kidney and brain.

When there is elevated blood pressure vascular resistance has to increase to maintain constant flow of blood. Because blood flow depends on pressure and resistance.

$$\text{Blood flow} = \frac{\text{Pressure across the vessel}}{\text{resistance offered by the vessel}}$$

When there is volume expansion initial response consists of elevated blood pressure due to increased cardiac output. But later peripheral resistance increases

gradually over time so that cardiac output comes back to normal. The effect of sodium on blood pressure is basically due to the provision of sodium with chloride, Nonchloride sodium salts have little or no role in causing elevated blood pressure.

When arterial blood pressure increases due to increased sodium chloride intake, urinary sodium excretion also tends to increase. Sodium balance is maintained by an increase in arterial blood pressure. Minute increases in the glomerular filtration rate, reduced absorbing capacity of the renal tubule to absorb sodium, and hormonal factors such as atrial natriuretic factor may be involved in causing pressure natriuresis. Greater increases in blood pressure are needed to maintain sodium balance.

There can be other mechanisms involved in this sodium chloride dependant hypertension. Kidneys may fail to excrete sodium because of intrinsic kidney disease, increased formation of mineralocorticoid hormone which retains salt by tubular re absorption. Increased neural activity to the kidney also increases reabsorption of sodium by the kidneys. In all these situations, a higher arterial blood pressure is required

to maintain sodium balance. There are certain disorders where salt wasting will be seen. It is found that such disorders are associated with low arterial blood pressure.

Hypertension seen in the End Stage Renal Disease(ESRD) is volume-dependent hypertension. In ~80% of ESRD patients, it is found that dialysis controls vascular volume and hypertension. In the rest 20%, it is found that rennin angiotensin system is overactive and contribute to hypertension, such patients may benefit from pharmacotherapy.

Autonomic Nervous System

The autonomic nervous system also plays an important role in regulating arterial blood pressure. It is mainly involved in regulating cardiovascular homeostasis. It regulates cardiovascular homeostasis with the help of volume, pressure, and chemoreceptor signals.

Adrenergic reflexes are known to modulate blood pressure over the short term. Adrenergic function, in concert with volume-related factors and hormonal factors, cause increased blood pressure over long term. The three major endogeneous catecholamines are nor epinephrine,

epinephrine, and dopamine. All the three play important roles in tonic and phasic cardiovascular regulation in the human body.

Guanosine nucleotide-binding regulatory proteins (G proteins) and intracellular second messengers are found to be the mediators of the activity of adrenergic receptors.. The receptor sites are specific both for the transmitter substance and function . Norepinephrine and epinephrine are agonists for adrenergic receptor subtypes with varying affinities.

Adrenergic receptors have been divided into two principal types: α and β. These types have been differentiated into α_1, α_2, β_1, and β_2 receptors. α_1 receptors are present on postsynaptic cells in smooth muscle. These receptors are known to cause vasoconstriction. α_2 Receptors are present on presynaptic membranes of postganglionic nerve terminals which are known to synthesize norepinephrine.

When activated by catecholamines, $\alpha_2$receptors act as negative feedback controllers. They inhibit the release of norepinephrine. α_1-adrenergic receptors enhances renal tubular sodium re absorption

.Activation of β_1 receptors in myocardium stimulates the rate and strength of cardiac contraction. It increases the cardiac output. β_1 Receptor activation also results in renin release from the kidney. Circulating catecholamine concentrations may affect the number of adrenoreceptors in various tissues.

Several reflexes are found to be involved in the regulation of the blood pressure on a minute-to-minute basis. Arterial baroreflex is mediated by stretch-sensitive sensory nerve endings. These receptors are located in the carotid sinuses and the aortic arch. The firing rate of these baroreceptors are found to be increased with increasing arterial blood pressure. The net effect is a decrease in sympathetic outflow. This results in reduction in heart rate s well as arterial pressure.

Acute fluctuations of blood pressure occurs during postural changes, physiologic or behavioral stress, and blood volume changes. In such acute situations baroreceptors play a vital role in BP regulation. However, the baroreflex activity declines overtime. It adapts to sustained increases in arterial blood pressure so that the baroreceptors are reset to higher blood pressures. Patients with autonomic neuropathy and altered

baroreflex function are found to experience labile blood pressures. They will have fluctuations in arterial blood pressure with the difficult-to-control episodic pressure spikes associated with tachycardia.

Increased sympathetic outflow is associated with hypertension both in normal as well as obese individuals. It is found that sympathetic outflow is more in hypertensives than in normotensives. This based on the recordings of postganglionic muscle nerve activity. Sympathetic outflow is more in obesity-related hypertension as well as in obstructive sleep apnea.

Baroreceptor activation via electrical stimulation of carotid sinus has been shown to reduce blood pressure in patients with "resistant" hypertension. Drugs blocking the sympathetic nervous system are potent antihypertensives. This indicates that the sympathetic nervous system plays a permissive role in the maintenance of increased arterial blood pressure. Pheochromocytoma is an example of hypertension related to increased catecholamine production

Renin-Angiotensin-Aldosterone system

Renin is a substance secreted by juxtaglomerular cells in the kidney in response to the low blood volume. Renin is an aspartyl protease that is synthesized as an enzymatically inactive precursor, prorenin. Prorenin may be secreted directly into the circulation or it may be activated within secretory cells and released as active form of renin. Although human plasma contains two to five times more prorenin than renin, there is no evidence that prorenin contributes to the physiologic activity of this system. It converts angiotensinogen secreted by liver into angiotensin I. Angiotensin I is converted into angiotensin II in the lungs by angiotensin converting enzyme.

Angiotensin II is a potent constrictor of blood vessels. So it increases blood pressure. It also stimulates adrenal cortex to secrete aldosterone. This increases absoption of sodium and water in kidneys. This results in volume expansion and hence, increase in arterial blood pressure;

There are three primary stimuli for renin secretion:

1. Decreased NaCl transport in the distal portion of the thick ascending limb of the loop of Henle that abuts the corresponding afferent arteriole (macula densa),
2. Decreased pressure or stretch within the renal afferent arteriole (baroreceptor mechanism)
3. Sympathetic nervous system stimulation of renin-secreting cells.

Conversely, NaCl transport in the thick ascending limb of loop of Henle inhibits rennin secretion. This effect is mainly by increased stretch within the renal afferent arteriole and by α_1 receptor blockade. In addition, angiotensin II directly inhibits renin secretion due to angiotensin II type 1 receptors on juxtaglomerular cells. It is found that renin secretion increases in response to pharmacologic blockade of either ACE or angiotensin II receptors.

Once released into the circulation, active renin cleaves a substrate, angiotensinogen, to form an inactive decapeptide, angiotensin I. A converting enzyme, located primarily but not exclusively in the pulmonary circulation, converts angiotensin I to the angiotensinII which

is an octapeptide. The same converting enzyme cleaves a number of other peptides also. This includes vasodilator substance bradykinin which is inactivated.

Angiotensin II primarily acts through angotensin II type 1 (AT_1) receptors on cell membranes. Angiotensin II is a potent pressor substance. It is the primary tropic factor responsible for the secretion of aldosterone by the zona glomerulosa of the adrenal cortex. Angiotensin II is also a potent mitogen .It stimulates vascular smooth muscle cell and myocyte growth.

Angiotensin II may play a role in the pathogenesis of atherosclerosis apart from its hemodynamic effects. It has a direct cellular action on the vessel wall.

An angiotensin II type 2 (AT_2) receptor has been recognized. It is widely distributed in the kidney. It has the opposite functional effects of the AT_1 receptor.

The AT_2 receptor induces vasodilation. It causes sodium excretion. It inhibits cell growth and matrix formation. Experimental evidence suggests that the AT_2 receptor improves vascular remodeling .This effect is mainly by stimulating smooth muscle cell apoptosis. This contributes to the regulation of glomerular filtration rate. AT_1 receptor blockade induces an increase in AT_2 receptor activity.

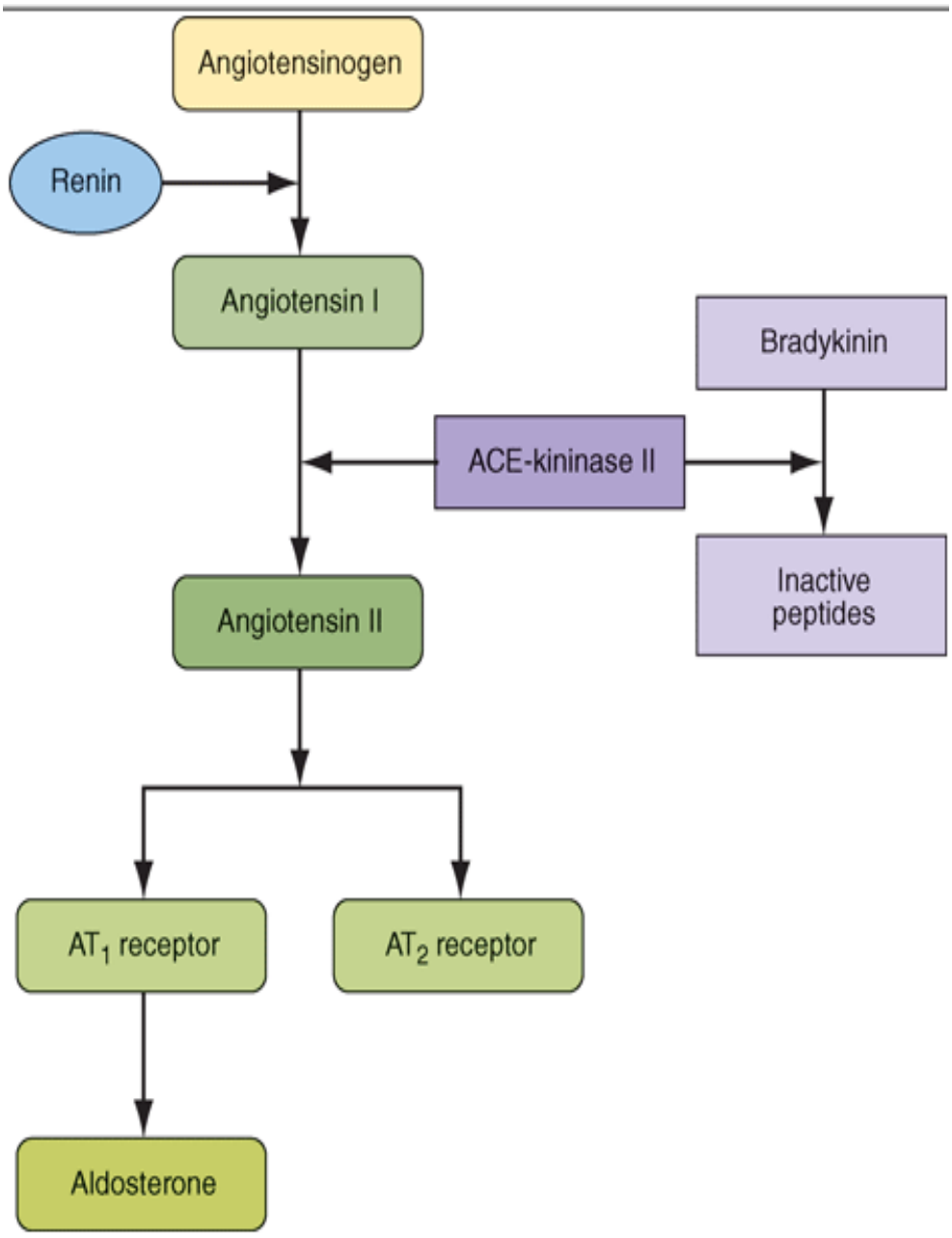


Fig 1: Renin angiotensin aldosterone system

Renin-dependent hypertension is exemplified by tumours secreting rennin. Benign hemangiopericytomas of the kidney, wilms tumours, and carcinomas of the kidney are all included. Renin is also produced by carcinomas of pancreas, liver , colon, lung and adrenals. In these cases apart from tumour resection therapy of hypertension also includes blocking the angiotensin receptors..

Renin also acts as a mediator of renovascular hypertension. In this scenario renal artery will be obstructed. This will result in decrease in perfusion pressure in the kidney. This leads to secretion of renin. This type of hypertension may become less dependent on renin as time passes due to the secondary damage to the renal tissue.

Renin, and angiotensin II are also synthesized locally in many tissues, including the brain, pituitary, aorta, arteries, heart, adrenal glands, kidneys, adipocytes, leukocytes, ovaries, testes, uterus, spleen, and skin. Angiotensin II in tissues may be formed by the enzymatic activity of renin or by other proteases, e.g., tonin, chymase, and cathepsins. In addition to regulating local blood flow, tissue angiotensin II is a mitogen that stimulates growth and contributes to modeling and repair. Excess tissue angiotensin II may contribute to atherosclerosis, cardiac hypertrophy, and renal failure and consequently may be a target for pharmacologic therapy to prevent target organ damage.

Angiotensin II is considered to be the primary tropic factor in synthesis as well as secretion of the mineralocorticoid, aldosterone by the adrenal cortex. Aldosterone activates amiloride-sensitive epithelial sodium channels which are present in principal cells of collecting ducts of the kidney. Thus increases sodium re absorption. To maintain electrical neutrality there will be exchange of potassium and hydrogen ions with sodium ions. It may result in hypokalemia and alkalosis.

The receptors for mineralocorticoid are also present in sweat glands, salivary glands and colon. Cortisol is also known to bind these receptors. But it is a less potent mineralocorticoid than aldosterone as it has to be converted to cortisone. Cortisone does not have affinity for the mineralocorticoid receptors.

Mineralocorticoiod-mediated hypertension is exemplified by primary aldosteronism. In this aldosterone synthesis is not under the control of rennin- angiotensin. Renin release is suppressed by increased volume.

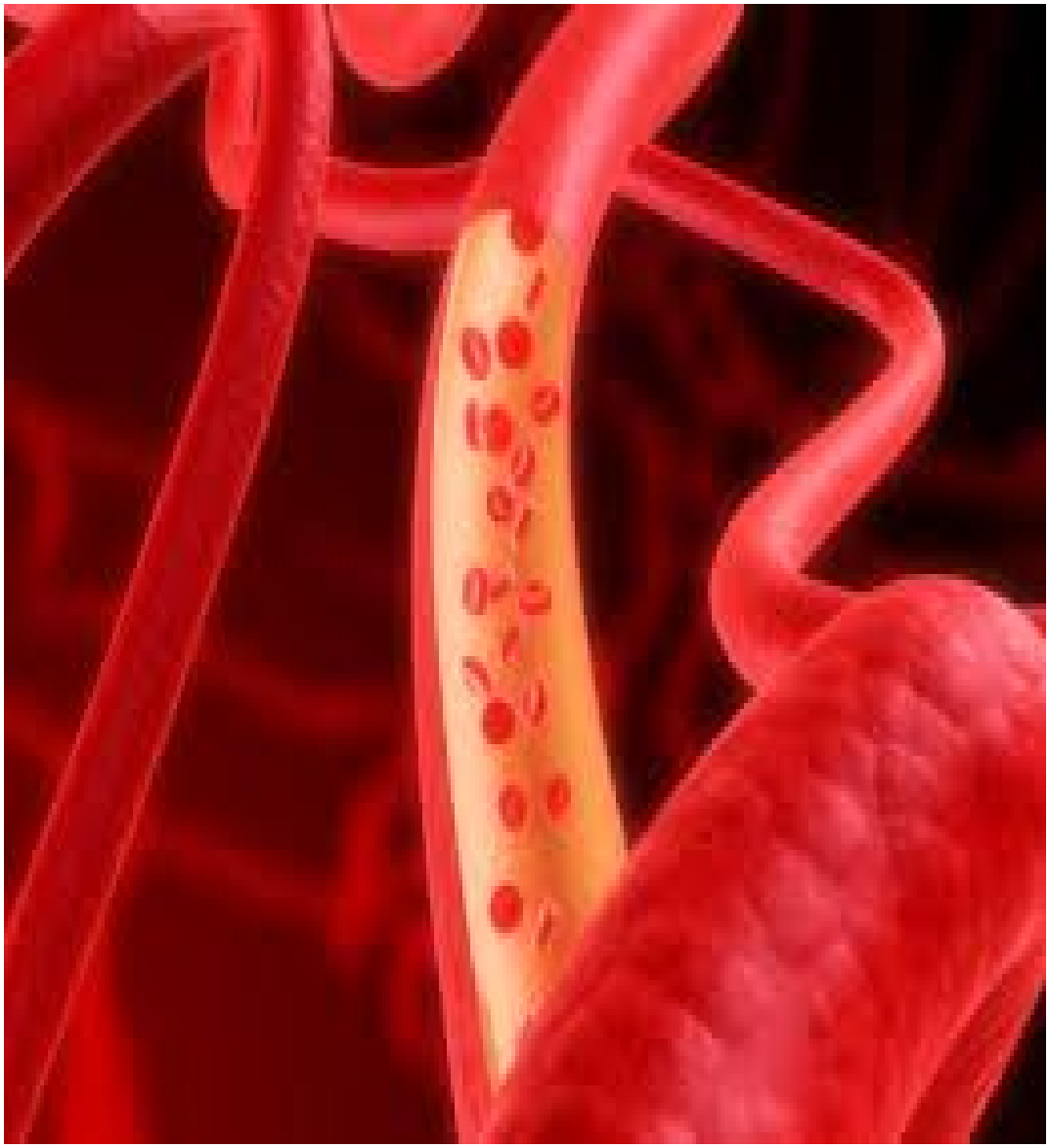
It is found that aldosterone also acts on non epithelial targets. Activation of mineralocorticoid receptor leads to alteration of structure and function of the kidney, heart and blood vessels. This leads to sclerosis of the nephrons, fibrosis of the myocardium and inflammation

of vessels due to oxidative stress. High salt intake is found to exacerbate these effects. These effects are amplified by a high salt intake.

Animal studies show that high aldosterone causes fibrosis of the myocardium and hypertrophy of the left ventricle. This myocardial fibrosis can be prevented by spironolactone which is an aldosterone antagonist. Left ventricular hypertrophy is seen in both essential hypertension as well as primary aldosteronism. Spironolactone in low doses reduces the risk of heart failure progression in patients with CHF. In the kidney aldosterone excess can lead to glomerular hyperfiltration and albuminuria. this effect is reversible with spironolactone and adrenelectomy.

Increased renin-angiotensin-aldosterone activity is not exclusive to hypertension. When there is low salt diet or contraction of plasma volume rennin-angiotensin activity increases to maintain the homeostasis. In cases of congestive heart failure and liver disease there can be secondary aldosteronism.

Vascular contribution to hypertension



Arterial pressure is determined by the compliance of arteries which contribute to the peripheral resistance and radius of the artery. Resistance offered by the arteries is inversely proportional to the fourth power of the arterial radius. So there will be significant increase in resistance with minute diminution in size of the lumen. In patients with hypertension there is reduction in diameter of the lumen of small arteries and arterioles with changes in functional, mechanical or structural aspects of these vessels.

Remodelling means there is alteration in the geometry of the vessel wall but there is no change in volume of the vessel. Vascular remodeling either hypertrophic or eutrophic increases the peripheral resistance by decreasing the size of the lumen. Low grade inflammation, fibrosis of the vessels and apoptosis are also involved in remodeling. Diameter of the lumen is also associated with the elasticity of the vessel wall. Vessels having high degree of vessel wall elasticity accommodate large volume of blood with small change in pressure where as vessels which are semirigid accommodate small increases in blood volume with greater increase in pressure.

Arteries are stiff in patients with hypertension. Vascular structural changes cause decreased vascular compliance. This results in elevated systolic pressure and widened pulse pressure. Stiffness of the arteries has been shown to have definite independent predictive value in causing cardiovascular disease. Usg and MRI are used for evaluating arterial stiffness.

Abnorrmalities of vascular growth and vascular tone are regulated by intracellular pH. Any alteration in pH changes the ion transport by the smooth muscle cells leading to the alterations in the vascular tone and contributes to hypertension.

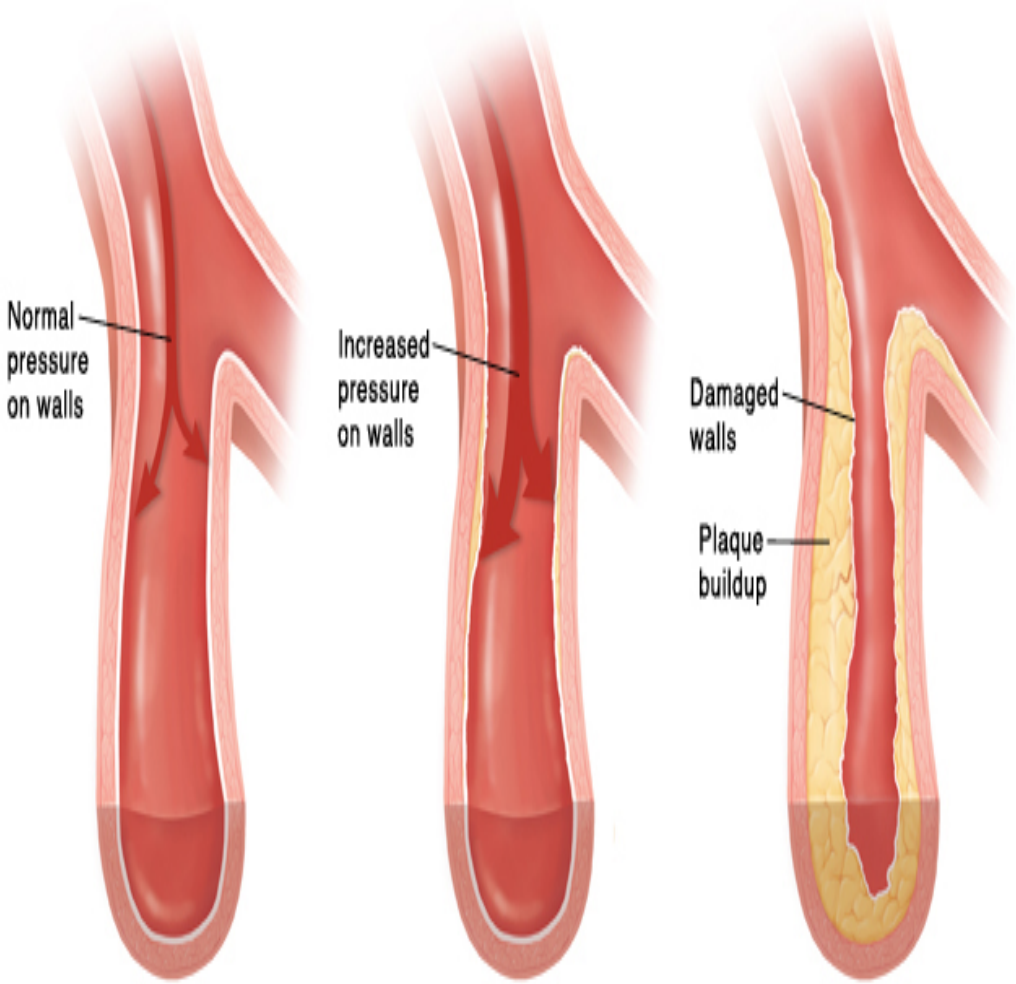
Three main transport systems are (1) sodium($Na^{+)}$-hydrogen $(H)^{+}$ exchange, (2)Sodium($Na^{+)}$-dependent bicarbonate($HCO_3^{-)}$-chloride(Cl^{-}) exchange, and (3) cation-independent bicarbonate$(HCO_3)^{-}$ chloride(Cl^{-}) exchange. Na^{+}-H^{+} exchanger activity is enhanced in hypertension. This increases vascular tone .

Two mechanisms are involved in this process. 1) when sodium entry increases it activates Na^{+}-Ca^{2+} exchange. This results in increased intracellular calcium. 2) Elevated pH enhances calcium sensitivity of the vessel wall contractile apparatus leading to an increase in contractility for a given calcium concentration. Increased Na^{+}-H^{+} exchange increases

sensitivity to mitogens leading to increased growth of vascular smooth muscle cells.

Vascular tone is also modulated by endothelial function. Endothelial cells synthesize and secrete vasoactive substances including nitrous oxide. Nitrous oxide palys a major role in vasodilatation. This kind of vasodialatation which is endothelium dependant is impaired in patients with hypertension. Vascular endothelium produces a substance called endothelin.It is a vasoconstrictor peptide. It is shown that endothelin antagonists do help in treating resistant hypertension. It is not known whether vascular abnormalities of ion transport related to hypertension and endothelial dysfunction seen in hypertension are primary alterations or secondary consequences of elevated BP. There is little evidence that weight loss, aerobic exercise and antihypertensive agents help in improving vascular compliance and endothelial function.

Pathologic Consequences of Hypertension



Hypertension is an independent predisposing factor for heart failure, coronary artery disease, stroke, renal disease, and peripheral arterial disease (PAD).

Heart

Hypertension is considered to be an independent risk factor for major health issues. Heart disease is the most common cause of death in hypertensive patients.

Hypertensive heart disease is the result of structural and functional adaptations leading to left ventricular hypertrophy, CHF, abnormalities of blood flow due to atherosclerotic coronary artery disease and microvascular disease, and cardiac arrhythmia.

Both genetic as well as hemodynamic factors contribute to left ventricular hypertrophy.

Electrocardiography and Echocardiography will help in diagnosing left ventricular hypertrophy. Echocardiography also measures the wall thickness of the left ventricle. Incresed wall thickeness is a risk factor for coronary artery disease, stroke, congestive heart failure and sudden death. It is shown that strict control of BP can regress or reverse this hypertrophy. It is important to reverse or regress this hypertrophy of the left ventricle and reduce the risk of cardiovascular disease.

It is known that congestive heart failure occurs when there is systolic dysfunction, diastolic dysfunction or in combined systolic and diastolic dysfunction. Diastolic dysfunction ranges from asymptomatic cardiac disease to full blown heart failure in patients with hypertension. One third of patients with congestive heart failure have diastolic dysfunction and normal systolic function. Hypertensive heart disease causes early diastolic dysfunction which is exacerbated by the hypertrophied left ventricle and ischemia.

Brain

Cerebrovascular disease is considerd as the second most frequent cause of death worldwide. Hypertension is the strongest risk factor for the development of stroke. It is estimated that 85% of strokes are ischemic and remainder are haemorragic. Stoke incidence increases progressively with increasing blood pressure levels, that too with systolic blood pressures in patients older than 65 years. Adequate control of hypertension is shown to reduce the incidence of both ischemic as well as haemorragic stroke.

Hypertension is known to cause impairement in cognition in older individuals. Longitudinal studies have shown that there is an association between hypertension during midlife and cognitive impairment in late life. Cognitive decline may be due to single large infarct or due to multiple lacunar infarcts. There can be occlusive small vessel disease causing ischemis of subcortical white matter leading to cognitive dysfunction.

Cerebral blood flow has autoregultiion. In patients with malignant hypertension there is impairment of autoregulaton and patients can develop encephalopathy. Signs and symtomes are severe headache, projectile vomiting, seizures, focal neurological deficits,altered mental status ,stupor and coma.

Kidney

The kidney is considered to be a target as well as source of hypertension. Renal disease is the most common cause for secondary hypertension. Failure to excrete sodium, increased secretion of renin and overactive sympathetic system are all proposed mechanisms in causing hypertension. Hypertension is a risk factor for renal injury and end stage renal disease. Risk association is more with the systolic than the diastolic blood pressure. Proteinuria is a marker for hypertension induced renal

damage. It asseses the severity and predicts the progression of the disease.

Hypertension causes atherosclerosis in preglomerular arterioles. This leads to ischemia in glomeruli and post glomerulr structures. There is loss of autoregulation of blood flow to the kidney. This results in lowering of threshold in causing renal damage. This result in a a vicious cycle.

The renal damage results in nephron loss resulting in severe hypertension, glomerular hyperfiltration, and increased damage to the kidney. This will lead to glomerular sclerosis. The renal tubules ultimately become atrophic due to ischemia. Glomerular pathology progresses to glomerulosclerosis, and eventually the renal tubules may also become ischemic and gradually atrophic. There will be fibrinoid necrosis of the afferent arterioles and focal necrosis of tuft of glomerulus in malignant hypertension. So it is important to detect renal injury in the early stages.

Peripheral Arteries

Though blood vessels contribute to the development of hypertension they are also the target organs for the atherosclerotic disease resulting from a chronically elevated arterial pressure. As this study is about ankle brachial index which is used to detect peripheral artery disease, this particular complication is discussed in detail.

PERIPHERAL ARTERY DISEASE

Arterial occlusive diseases constitute the leading overall cause of death in developing as well as developed countries. Adverse life threatening events are due to impaired circulation to major end organs like heart, brain, abdominal viscera or extremities. In addition to death usually caused by myocardial infarction or stroke, significant disability and loss of function are troublesome to the society. Atherosclerosis is the main pathology behind this arterial occlusive disease.

Atherosclerosis

Atherosclerosis is the main pathology behind major catastrophic events like myocardial infarction and stroke. As stated earlier vessels are the target organs for increased blood pressure. Alteration in the endothelial function, turbulence of blood flow, increased lipids all contribute to the development of this pathology.

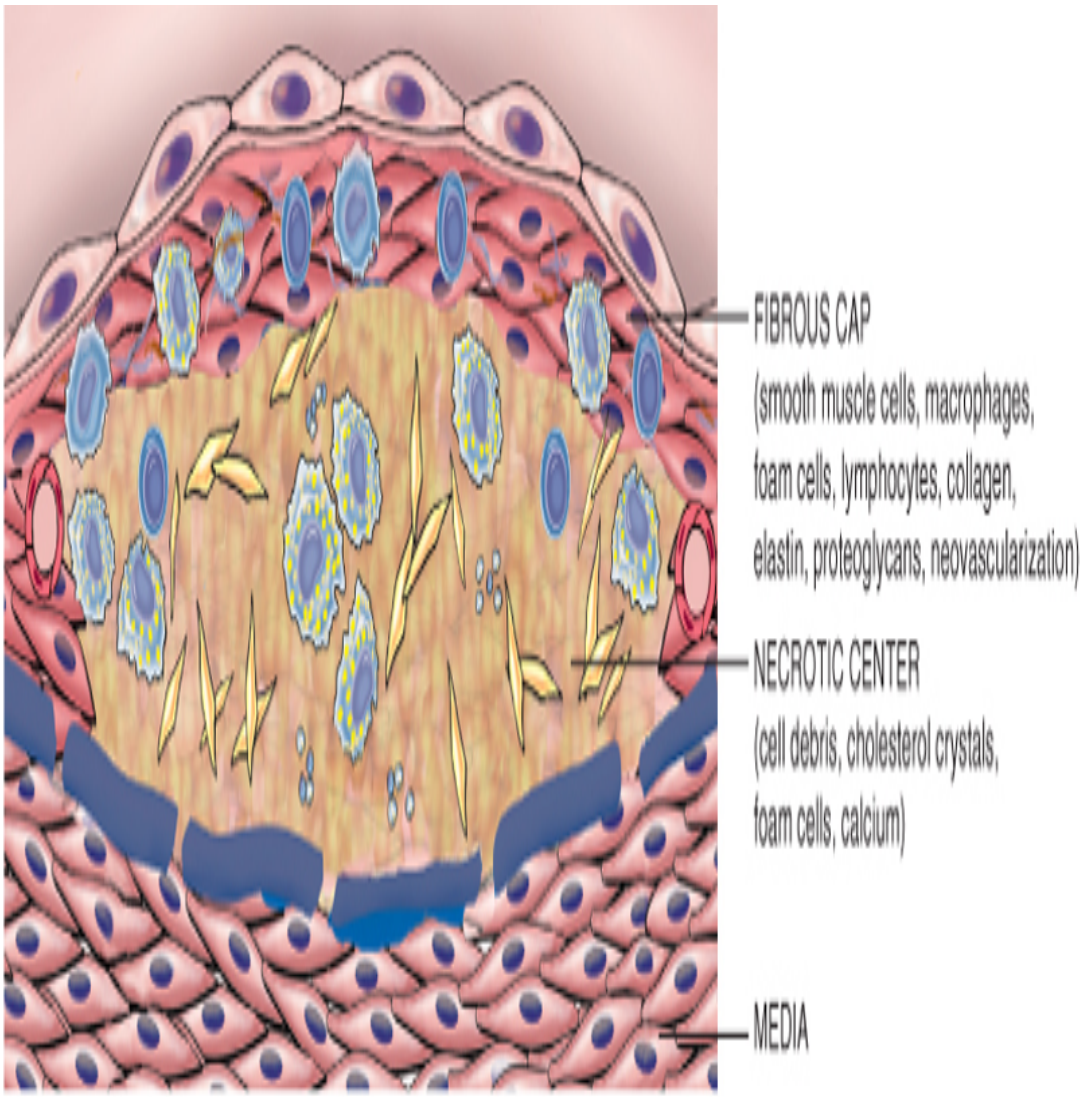


Fig 2: Atherosclerotic plaque

atherosclerosis is a complex, chronic inflammatory process affecting the elastic and muscular arteries. It is both systemic and segmental, with clear predilections for certain locations in arteries and relative sparing of others. The earliest lesions (fatty streaks) may be detected as early as in childhood in susceptible individuals. An atheromatous plaque will have a raised lesion with a soft, yellow, grumouscore of lipid ,mainly cholesterol and cholesterol esters, which is covered by a firm, white fibrous cap . Besides obstructing blood flow, these plaques weaken the underlying media and can rupture by themselves causing acute catastrophic vessel thrombosis.

Before the clinical manifestations develop lesions progress through well-characterized pathologic stages. Population-based studies have demonstrated important risk factors. The most important independent risk factors are hypertension, hypercholesterolemia, smoking, and diabetes mellitus. Other risk factors are depicted in the following table.

Risk Factors for Atherosclerosis

Major Risks	Lesser, Uncertain, or Nonquantitated Risks
Nonmodifiable	Obesity
Increasing age	Physical inactivity
Male gender	Stress ("type A personality)
Family history	Postmenopausal estrogen deficiency
Genetic abnormalities	High carbohydrate intake
	Lipoprotein(a)
Potentially Controllable	Hardened (trans)unsaturated fat intake
Hyperlipidemia	
Hypertension	*Chlamydia pneumoniae infection*
Cigarette smoking	
Diabetes	
C-reactive protein	

Table1: Risk factors for atherosclerosis

Risk factors

Firmly Established
Hypertension Cigarette smoking Hypercholesterolemia Diabetes mellitus
Relative
Advanced age Male gender Hyperhomocysteinemia Hypertriglyceridemia Sedentary lifestyle Family history

Table 2: Firmly established and relative risk factors

Hypercholesterolemia is associated with increased risk. Of prognostic significance is the relative apportioning between the subclasses of lipoproteins: the atherogenic, low-density fraction (LDL)and the athero protective high-density lipoprotein fraction (HDL). Studies have proved a strong positive correlation between cardiovascular disease secondary to atherosclerosis and elevated total and LDL cholesterol and strong negative correlation with HDL levels.

Hypertension is important independent risk factor for coronary atherosclerosis. With a continuous increase in relative risk associated with each increment of pressure there will be a continuous increase in relative risk for coronary atherosclerosis. Hypertension alone can increase the risk of IHD by approximately 60% in comparison with normotensive populations. Left ventricular hypertrophy probably represents a marker of long-standing functional hypertension .

Cigarette smoking is strongly associated with the incidence of atherosclerosis. The mechanism is likely to be due to direct toxicity of tobacco metabolites on the vascular endothelium, mainly by creating oxidant stress.

Diabetic patients are at markedly increased risk for atherosclerosis, leading to higher rates of myocardial events, stroke, and amputation.

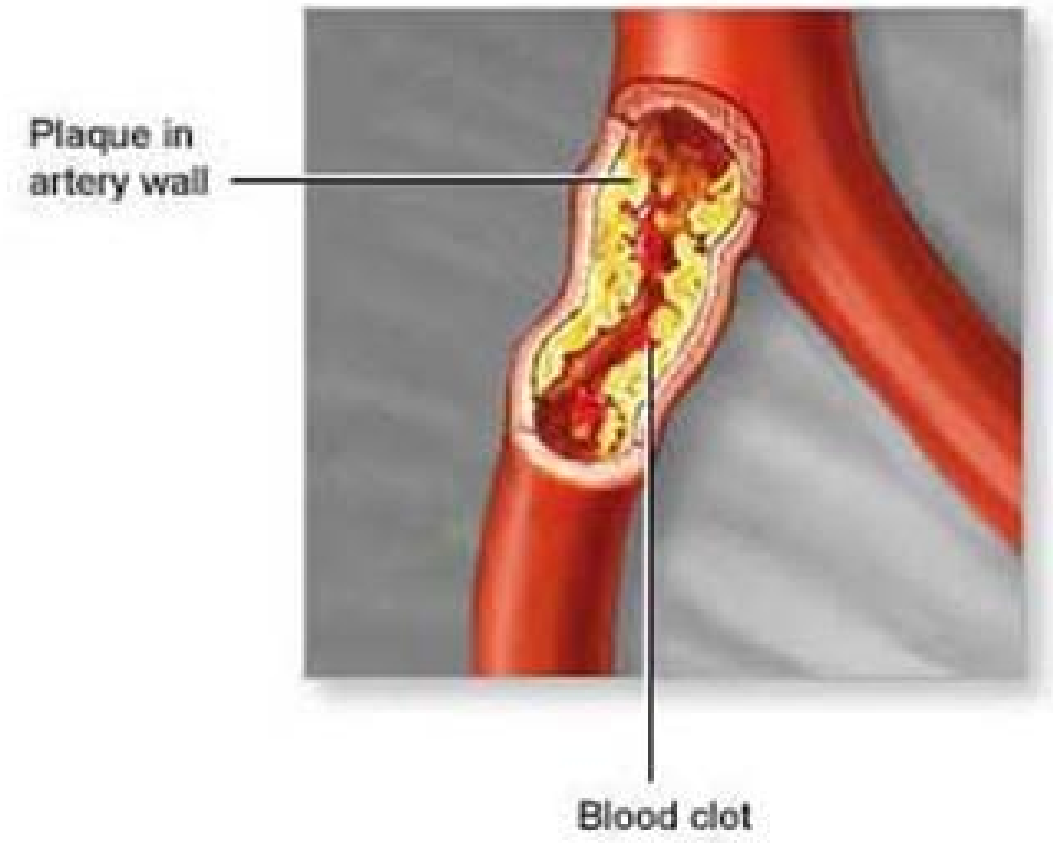
Age and gender also have an important influence. Prevalence increases with the advancing age. The increased risk is associated with male gender and postmenopausal states in women.

There are Guidelines for Risk Factor Modification. The following table highlights the guidelines.

Lipid Management
Goal: Primary—serum LDL <100 mg/dL; secondary—HDL >35 mg/dL, TG <200 mg/dL *Approach:* Diet: <30% fat, <7% saturated fat, <200 mg/day cholesterol; specific drug therapy targeted to lipid profile
Weight Reduction
Goal:<120% of ideal body weight *Approach:* Physical activity, diet as outlined
Smoking
Goal: Complete cessation *Approach:* Behavior modification, counseling, nicotine analogues
Blood Pressure
Goal:<140/90 mm Hg *Approach:* Weight control, physical activity, sodium restriction, antihypertensive drugs
Physical Activity
Goal: At least 30 minutes of moderate exercise 3 to 4 times per week *Approach:* Walking, cycling, jogging, lifestyle and work activities

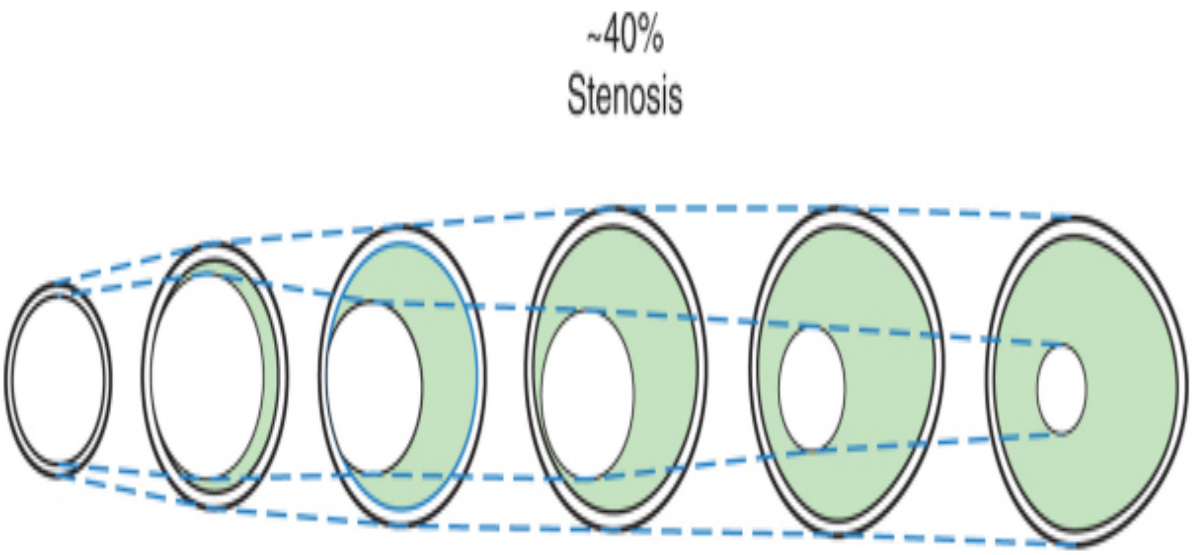
Table 3

PATHOLOGY AND THEORIES OF ATHEROGENESIS



Atherosclerotic plaque is the pathologic hallmark of atherosclerosis. Major components of plaque:connective tissue (matrix), smooth muscle cells, lipid, and inflammatory cells (predominantly macrophages)TT. The presence of lipid within these lesions is a prominent feature. An important concept linking plaque morphology with clinical events is the relationship between the fibrous cap and the underlying necrotic lipid core.[26] The contents of this central region are thrombogenic when exposed to circulating blood, when a thin fibrous cap ruptures or ulcerates. This phenomenon is considered to be an important mechanism by which lesions of relatively mild hemodynamic significance may cause acute thrombosis and tissue infarction. It is clear that both the mechanical characteristics of the plaque and the degree of luminal stenosis are of clinical importance.

The anatomic distribution of atherosclerosis is remarkably constant. It is due to the important role played by the hemodynamic stresses.[26] Plaques have tendency to get concentrated at bifurcations or bends, where there are local alterations in shear stress, turbulence, flow separation and stasis are seen. The proximal coronary arteries, infrarenal abdominal aorta, iliofemoralarteries, carotid bifurcation, and popliteal arteries are commonly affected. Upper extremity vessels, renal, common carotid, and mesenteric arteries beyond their origins are usually spared.



Atherosclerotic plaques are dynamic lesions . They may undergo progression or regression over time. The underlying arterial wall also undergoes adaptive remodeling. Arterial enlargement is a well known feature of atherosclerosis. It will often result in relative preservation of the lumen until plaque volume reaches a threshold size (~40% stenosis) beyond which compensation will fail and lumen narrowing will become progressive[27] . Medial atrophy can also occur, in which mechanical stability of the wall is impaired. This may be one of the mechanism for aneurysmal disease.

The “response to injury” hypothesis and its recent modifications including the concept of endothelial cell dysfunction, is the leading theory of pathogenesis.[28] This hypothesis emphasizes the important roles for lipid, inflammation, and thrombosis in addition to proliferation and dysfunction of the cells in the arterial wall. In the earlier versions of this theory, a focal denuding injury to the endothelium was thought to be triggering event.

More recently it is known that endothelial injury includes a mechanically intact but phenotypically altered endothelial monolayer . The source of injury may include hemodynamic stress, hypoxia, toxic metabolites (e.g., cigarette smoke, homocysteine), or infectious agents (cytomegalovirus, *Chlamydia*, herpesvirus). The final pathway is a loss of the atheroprotective effects of normal endothelium, such as its barrier function, anti proliferative and potent anti adhesive properties.

The vascular smooth muscle cell (SMC) plays a central role in the formation of the lesion. Migration and proliferation of arterial medial SMCs result in a cellular neointima. The intimal SMC undergoes a change from a contractile to a secretory state and produces the extracellular matrix. Lipid accumulation in the vessel wall is an important early event. Oxidation of lipid, particularly LDL particles takes place. It produces metabolites which potentiate the “activated” endothelial

phenotype characterized by the expression of procoagulant (e.g., tissue factor) molecules, proinflammatory molecules (e.g., leukocyte adhesion molecules) and as well as producing a decrease in protective substances (e.g., nitric oxide).

Circulating monocytes are recruited by adhesion to activated damaged endothelium or to exposed matrix. They enter the wall to become macrophages, and scavenge lipid. T lymphocytes are also recruited, and together all these inflammatory cells secrete an array of cytokines (especially interleukin-1, transforming growth factor-b and tumor necrosis factor-a,), causing the inflammation. Macrophages are also important sources of matrix degrading enzymes involved in wall remodeling and plaque stability.

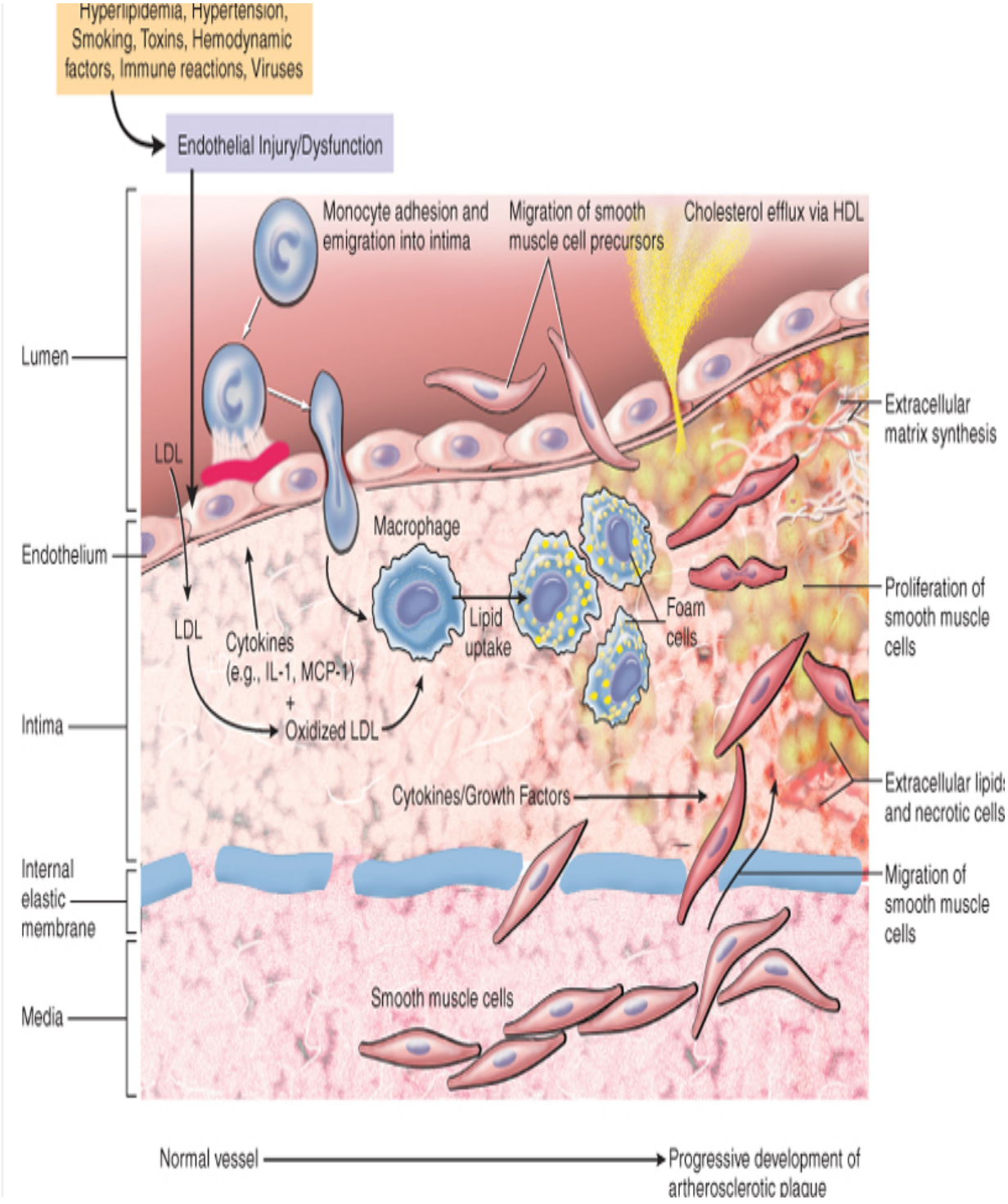


Fig 3: Pathogenesis off atherosclerosis

Platelets adhere to dysfunctional endothelium, exposed matrix, and monocytes-macrophages. The growth factor known as platelet-derived growth factor (PDGF) is an important and potent stimulator of both migration and proliferation of SMCs, It has been found in abundance in atherosclerotic plaques. Apart from platelets different isoforms of PDGF are also produced by endothelial cells and by SMCs. Locally produced growth factors like basic fibroblast growth factor (bFGF), are likely to play a role in the SMC hyperplasia . Amplification occurs by numerous potential positive feedback loops between cytokines and growth factors .

An alternative explanation is given by the "monoclonal hypothesis," which hinges on the observation that many atherosclerotic plaques contain a clonally expanded population of SMCs.[30] The developing plaque is considered as a benign SMC neoplasm with an alteration in SMC phenotype to the secretory state. But this theory fails to explain all the epidemiologic and pathologic features of atherosclerosis and is currently less favored.

CLINICAL FEATURES OF PERIPHERAL ARTERY DISEASE

Asymptomatic

There is substantial evidence that says that progression of underlying peripheral artery disease is same whether it is symptomatic or asymptomatic.

The risk of local deterioration, leading to limb ischemia is not related to presence or absence of symptoms of intermittent claudication. So it is vital to detect this subgroup of patients early so that protective and preventive measures can be applied to reduce adverse outcomes.

Intermittent claudication

It is pain in calf, thigh and buttocks while walking. The pain is described variably as aching, cramping, tightness or fatigue. This is relieved by rest.

Patients with this symptom continues to have functional disability. A quarter of these patients will not experience significant deterioration. This stabilization of symptoms may be due to the development of collaterals or metabolic adaptation by the muscle. 25% of patients with intermittent claudication will deteriorate in clinical stage. This is seen most frequently during the first year after the diagnosis (7-9%). After that only 2-3% experience deterioration of symptoms. This apparent

difference is because patients subjective perception of severity of claudication. When these patients are subjected to comprehensive functional status assessment their measured walking distance does decrease over the course of time. Only 1-3.3% of patients with intermittent claudication will require amputation over a 5 year period[31, 32].

A changing ABI predicts that patients disease is deteriorating. Studies have shown that patients with intermittent claudication who are in lowest strata of ankle brachial index have 8.5% risk of progression to severe ischemia or actual limb loss[33].

Critical limb ischemia

It refers to ischemic pain in the foot during rest with or without ulcers or gangrene. The estimated incidence ranges between 500-1000 new cases per million population per year. It is also a marker for generalized severe atherosclerosis. So these patients have poor prognosis .The primary goal of treating these patients is to relieve severe ischemic pain, prevent loss of the extremity, heal the ulcers, improve patients' quality of life and prolong survival.

Local physical examination findings

Peripheral arterial disease patients will have change in colour of the limb, change in temperature of the skin , decreased hair growth, atrophy of the muscles and hypertrophy of nails. Bruit may be heard over carotid artery, femoral artery or aorta . Absence of bruit does not rule out the presence of arterial disease. Ankle Brachial index, segmental pressure technique, Doppler study, transcutaneous oximetry, and arteriography are all used in diagnosing peripheral artery disease.

ANKLE BRACHIAL INDEX

Ankle Brachial Index:

The ankle brachial index a ratio. It has systolic pressure at the ankle as numerator and systolic pressure in the arm as denominator. The measurement is fast and quantification is easy. It is used to diagnose and evaluate peripheral artery disease.

Recently ABI is used to evaluate patients who are at increased risk for cardiovascular disease. An ABI < =0.9 has been shown to increase the risk of future myocardial infarction. This risk is related to the degree of reduction in ABI and it is independent of other known risk factors for cardiovascular disease.

ABI provides additional risk stratification in individuals with Framingham risk between 10-20% in 10 years. In this intermediate risk group abnormal ABI will move the patient to high risk group requiring secondary prevention strategy where as normal ABI will lower the risk requiring primary prevention strategy. It also indicates that peripheral vessels are having atherosclerosis even though patients are asymptomatic. In patients who have no symptoms reduced ABI is associated with reduction in functional capacity of limbs. This reduction in limb function is defined as decreased speed of walking and/or reduced walking distance observed during a six minute walk test.

Primary care medicine should include ABI measurement as a routine. In this context screening of individuals aged 50-69 years who are diabetics and hypertensives or screening all patients above seventy years showed prevalence of PAD as 29% in one study. ABI reporting requires a change of 0.15 in an isolated measurement or >0.10 if associated with change in patients' present clinical status , because in literature the reproducibility of ABI varies. The cut off point for diagnosing PAD is <=0.9 at rest[34].

Advantages of detecting reduced ABI:

a. Helps in confirming the diagnosis of PAD
b. Helps in detecting significant PAD in asymptomatic individuals
c. Helps in identifying patients with reduced limb function
d. As a prognostic indicator of cardiovascular disease.
e. Helps in risk stratification , with the lower spectrum having poor prognosis.

The normal ABI is >1.The cut off to diagnose PAD is <=0.9. Individuals with claudication are known to have ABI in the range of 0.5-0.7. Those who have rest pain are in 0.3-0.5 range. Patients with gangrene have ABI of less than 0.3. These ranges vary according to the degree of vessel all compressibility. The test is less reliable in people with heavily

calcified arteries. Due to non compressibility in diabetes and ESRD some patients may have ABI =1.4. These individuals need additional diagnostic tools for the assessment of the disease

ABI grading of peripheral artery disease

ABI	Disease grading	Disease severity
>1.3	0	Non compressible
0.95-1.30	0	Normal
0.60-0.94	1	Minimal
0.50-0.59	2	Mild
0.26-0.49	3	Mild to moderate
0.20-0.25	4	Moderate
0-0.19	5	Severe

Table : 4 ABI grading of peripheral artery disease

Experts believe that asymptomatic patients with low ABI should be treated similar to symptomatic patients as cardiovascular risk remains the same. A prospective study by name get ABI (German Epidemiological Trial on Ankle Brachial Index) showed that low ABI was associated with increased CVD risk and there is no statistically significant difference between asymptomatic and symptomatic groups.

Hence it is important to detect these patients with low ABI even when they are asymptomatic so that preventive measures can be applied to reduce the CVD risk. These include lifestyle modification, antiplatelet therapy, statins or ACE inhibitors.

AIM OF THE STUDY

- Aim of the study is to study the effect of hypertension on ankle brachial index.

- To assess the effect of following factors on ankle brachial index

- Age
- Sex
- Body Mass Index
- Duration of Hypertension

MATERIALS AND METHODS

SELECTION OF SUBJECTS:

Hypertensive patients who visit the Department of Medicine were enrolled for the study after excluding exclusion criteria.

Inclusion criteria :

Hypertensives according to jnc7.

Exclusion criteria :

- Patients who have diabetes mellitus
- Patients who have chronic kidney disease
- Patients who have coronary artery disease
- Patients who have dyslipidemia
- Patients who are smokers
- Patients with symptoms of peripheral artery disease

Setting : Kilpauk Medical College

Study design : Cross sectional study

Duration of study : 6 months

Sample size : 198

The data of each patient will be collected on a proforma specially designed for this study. This proforma includes

name,

age,

height of the patient,

weight of the patient,

systolic blood pressure of the patient

diastolic blood pressure of the patient

duration of hypertension

ankle brachial index.

This data will be analysed for statistical significance and correlation.

PROFORMA

NAME :

I.P/O.P.NO. :

AGE/SEX :

DATE :

OCCUPATION :

ADDRESS :

CONTACT NO :

COMPLAINTS :

HISTORY :

H/O HYPERTENSION DURATION :

SIGNIFICANT PAST HISTORY :

FAMILY HISTORY :

PERSONAL HISTORY :

TREATMENT HISTROY(DRUGS) :

GENERAL PHYSICAL EXAMINATION :

HT: WT: BMI:

VITALS:

BP: PR: RR:

SYSTEMIC EXAMINATION:

CVS:

RS:

P/A:

CNS:

Others

INVESTIGATIONS:

Ankle brachial index

RFT

Random blood sugar	Urea	Creatininie

ECG

ECHO

Lipid profile

Triglycerides	Total Cholesterol	HDL- C	LDL-C	VLDL-C

ANKLE BRACHIAL INDEX MEASUREMENT

Blood pressure was measured in both ankles and arms with the patient in supine position. Systolic blood Pressure was measured in both arms with the help of Blood pressure cuff and Doppler instrument (Ultra Tec PD1 v with a vascular probe of 5 MHz) in the ante cubital fossa. Systolic pressure was measured at the left and right dorsalispedis arteries. If it was not found in dorsalis pedis arteries, systolic blood pressure was recorded at the left and right posterior tibial arteries. The blood pressure cuff was applied to the ankle just proximal to the medial malleoli.

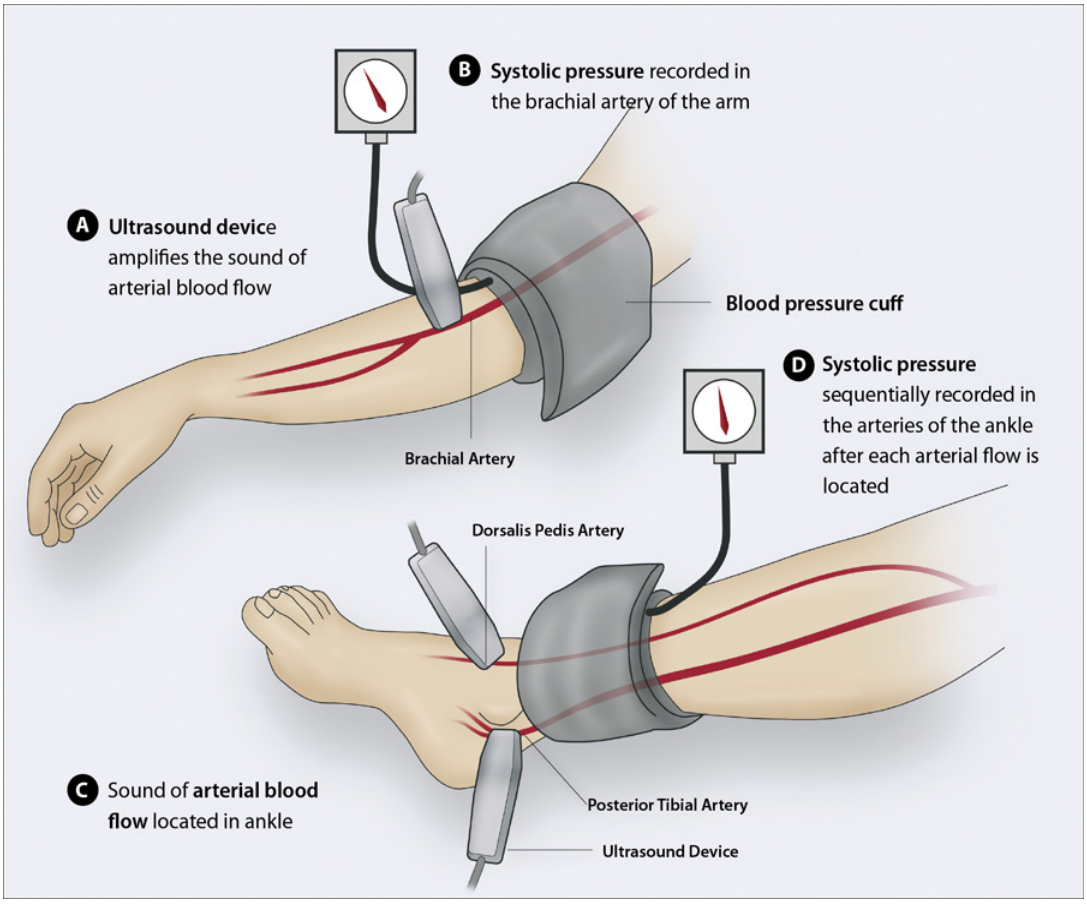


Fig 4 : ABI measurement

Blood pressure measurement

Blood pressure of the patient was measured with the help of mercury sphygmomanometer. It was measured after making patient relaxed for at least five minutes with the blood pressure cuff applied on the arm. It was measured in sitting position in both upper limbs, with the apparatus at the level of heart. The mean of the two readings taken at intervals of at least two minutes was used for data analysis. Pulse pressure was calculated by the formula: pulse pressure = systolic blood pressure - diastolic blood pressure.

Fig 5: Blood pressure measurement

Body Mass Index (BMI) measurement:

Body Mass Index was calculated with the help of the following formula

$$\text{Body mass index} = \frac{\text{Weight in Kg}}{\text{Height in m}^2}$$

Patients' weight was measured and it was expresses in kilogram.

Patients' height was measured and it was expressed in meters. Body mass index was calculated using the above formula.

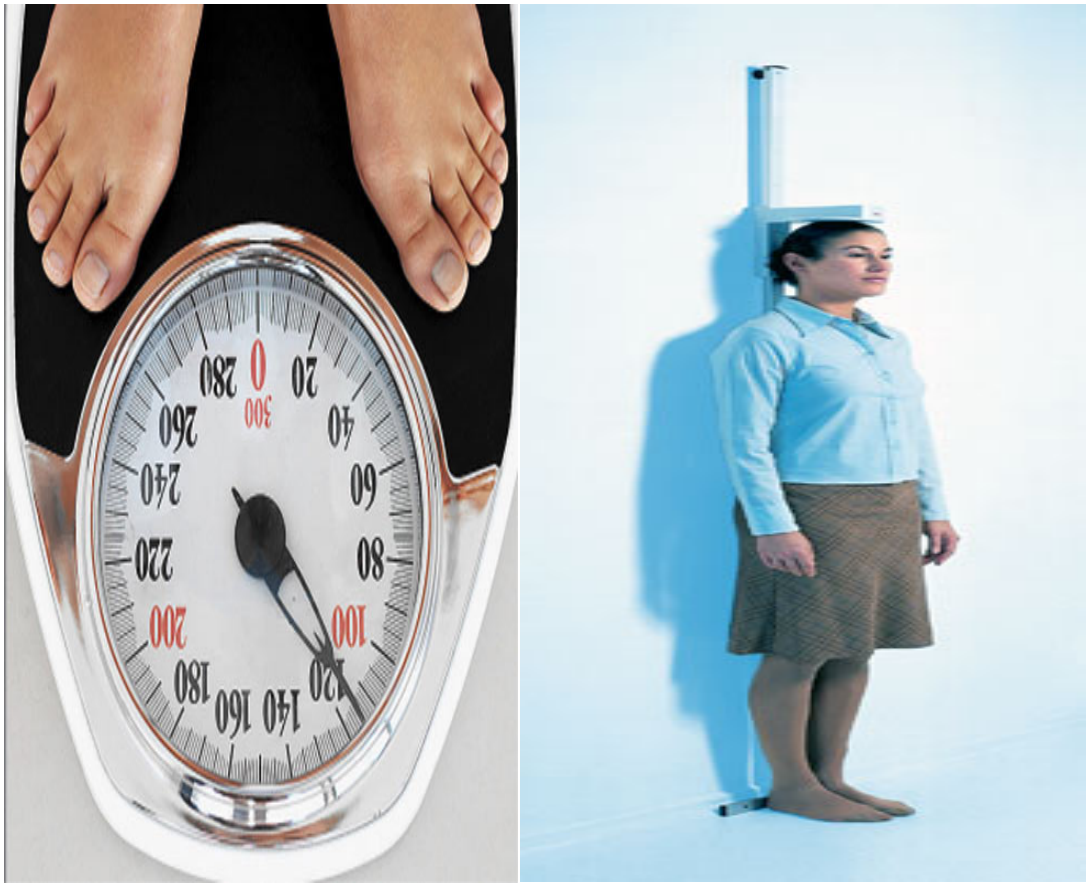


Fig 6: Weight and Height measurement

WHO classification of BMI	:	
Normal	:	18.5-24.9kgs
Overweight	:	25-29.9kgs
Obese	:	>30kgs

.

STATISTICAL ANALYSIS :

Data obtained were maintained in the master chart in Microsoft Excel format and analysis was done using SPSS 11.5 version. Data was arranged in proportions. The variables were compared using pearson correlation coefficient. A p value of <0.05 was considered to be statistically significant.

OBSERVATIONAL ANALYSIS

Age distribution in this study

Age in years	Number of subjects
21-30	7
31-40	29
41-50	68
51-60	67
61-70	23
71-80	2
81-90	2

Tables 5: Age distribution among the subjects

Distribution of Age

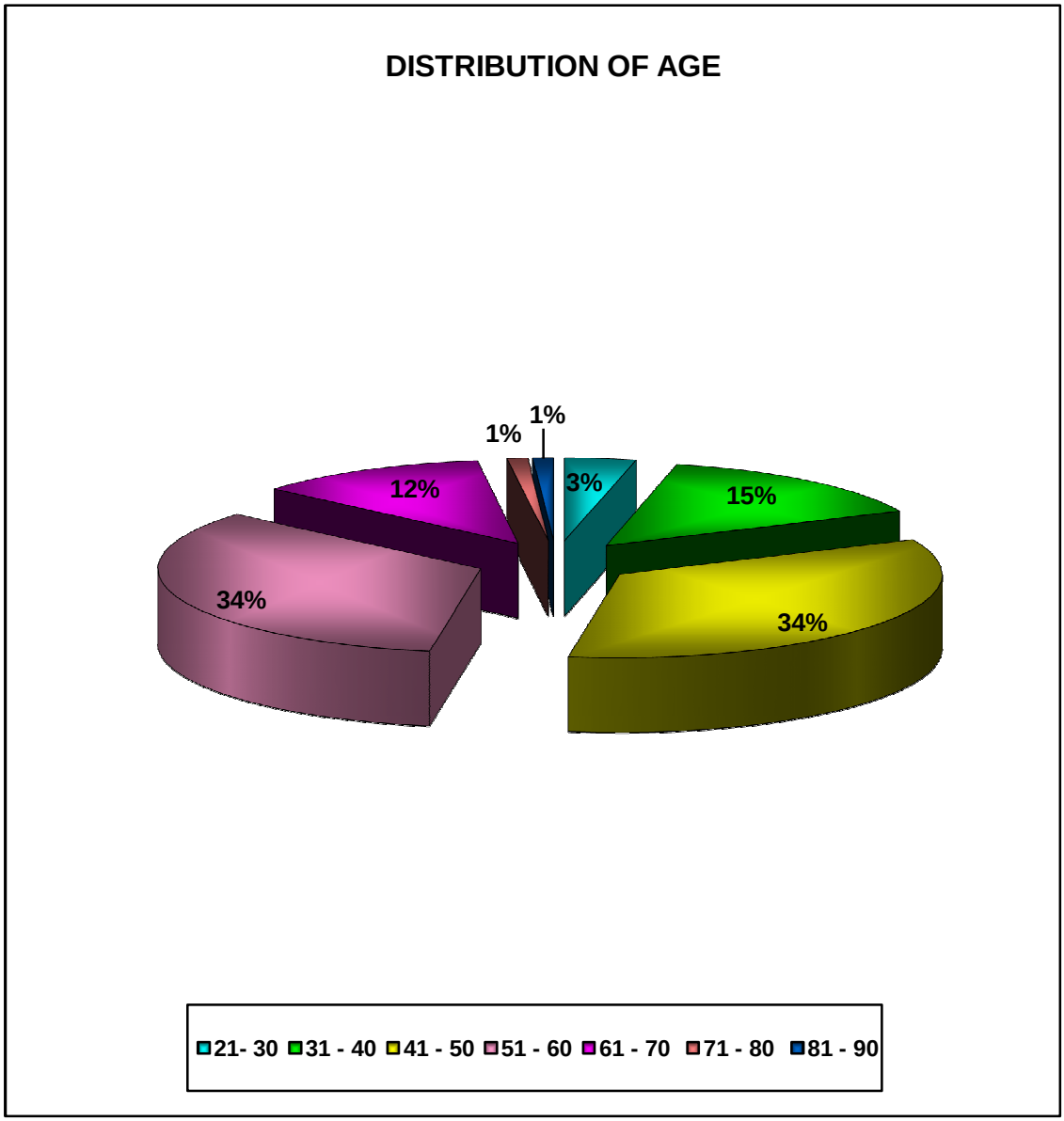


Fig 7: Distribution of age

A total of 198 patients were included in this study

- ✓ 3 % were in the age group of 21- 30 years;
- ✓ 15% were in the age group 31-40 years;34% were in the age group of 41-50 years;
- ✓ 34% were in the age group of 51-60 years;12% were in the age group of 61-70 years;
- ✓ 1% were in the age group of 71-80 years;
- ✓ 1 % were in the age group 81- 90 years.

Distribution of sex in this study

Male	62
Female	136

Table 6: Distribution of sex

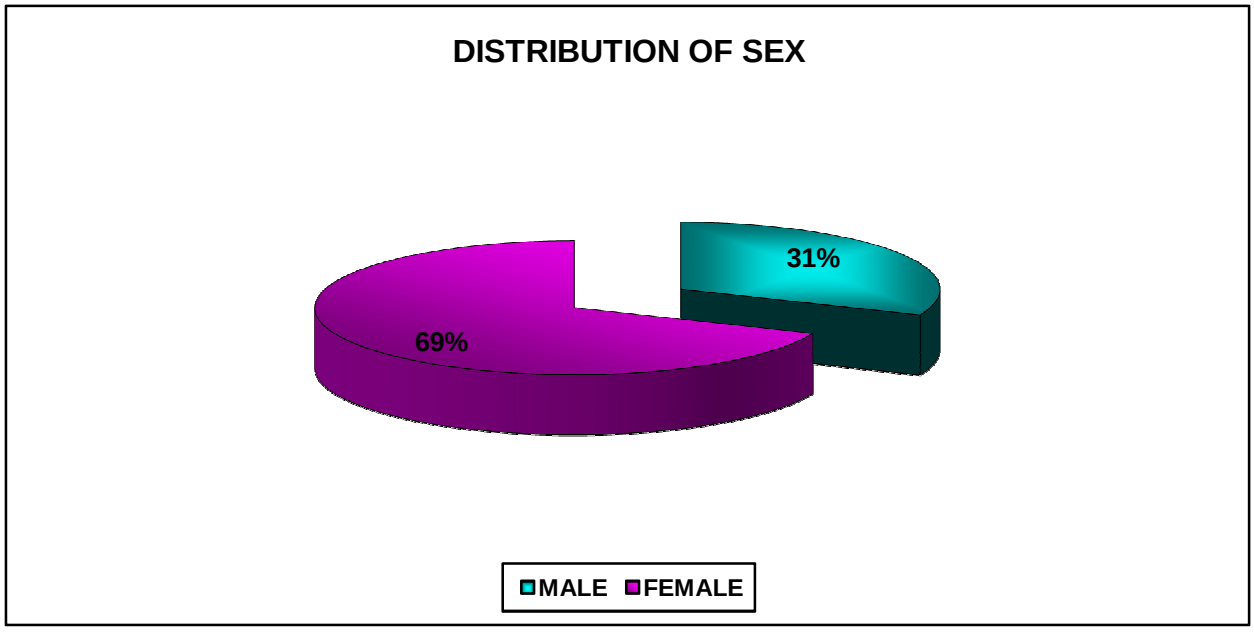


Fig 8: Distribution of sex

Out of 198 patients studied 136 were females(69%) and 32 were males(32%).

Distribution of BMI

NORMAL	104
OVERWEIGHT	89
OBESE	5

Table7 : Distribution of subjects according to BMI

This table depicts the BMI distribution in this study. This is plotted into a pie diagram for further simplification.

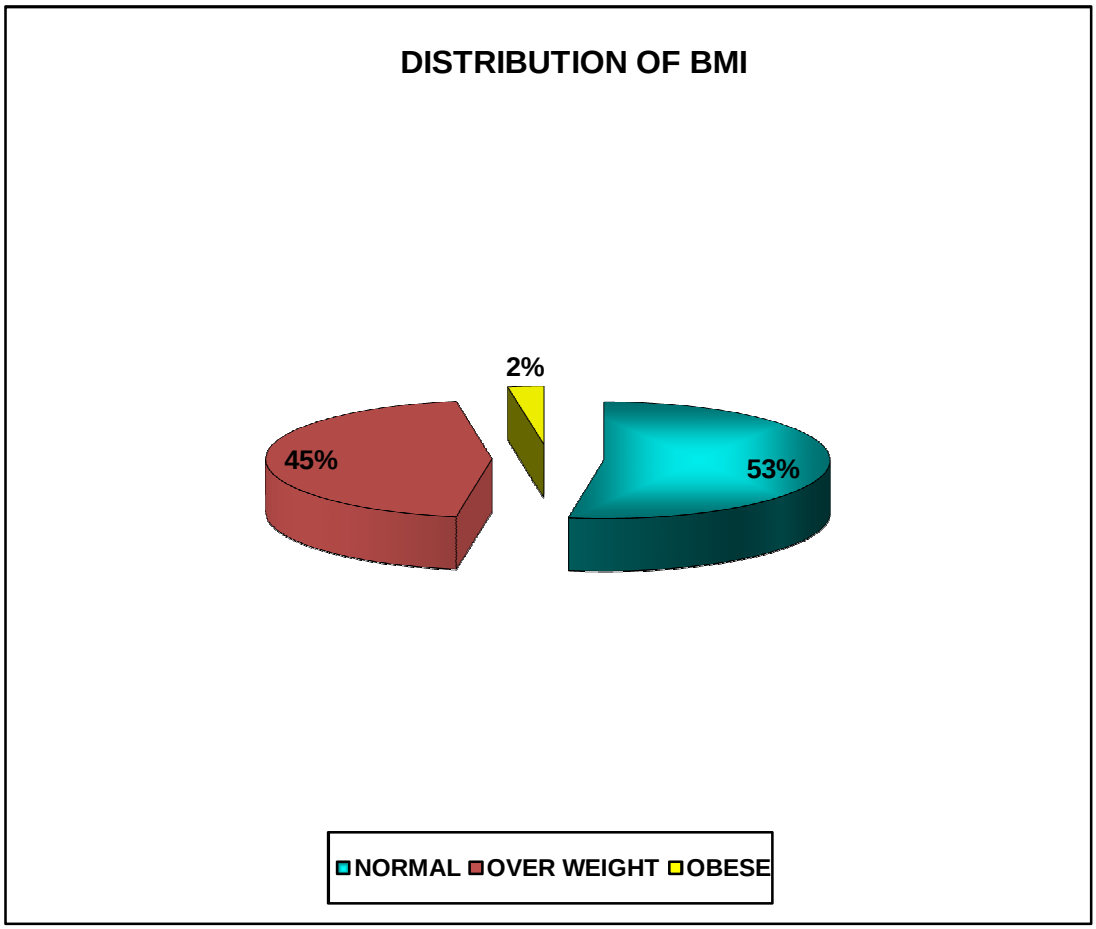


Fig 9: Distribution of BMI

In my study there were 53% patients with normal BMI. There were 45% of patients were overweight and 2% were obese

Distribution according to the duration of hypertension

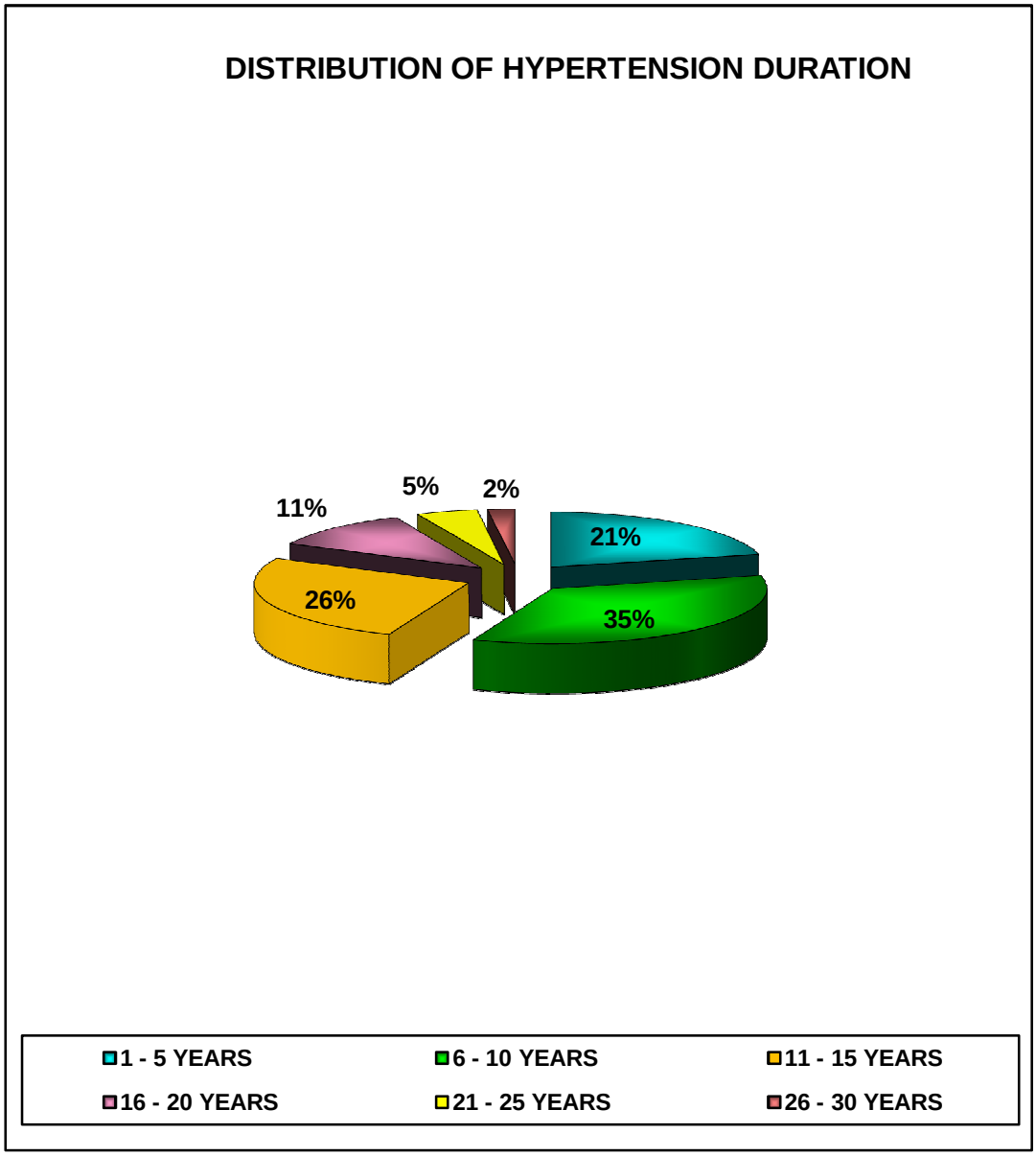


Fig 10: Distribution of Hypertension duration

Out of 198 patients,

- 42 patients (21%) were having hypertension for past 1-5 years;
- 69 patients (35%) were having hypertension for 6-10 years;
- 52 patients (26%) were having hypertension for 11-15 years;
- 22 patients (11%) were having hypertension for 16-20 years;
- 9 patients (5%) were having hypertension for 21-25 years
- 4 patients (2%) were having hypertension for 26-30 years.

Correlation between age and ABI

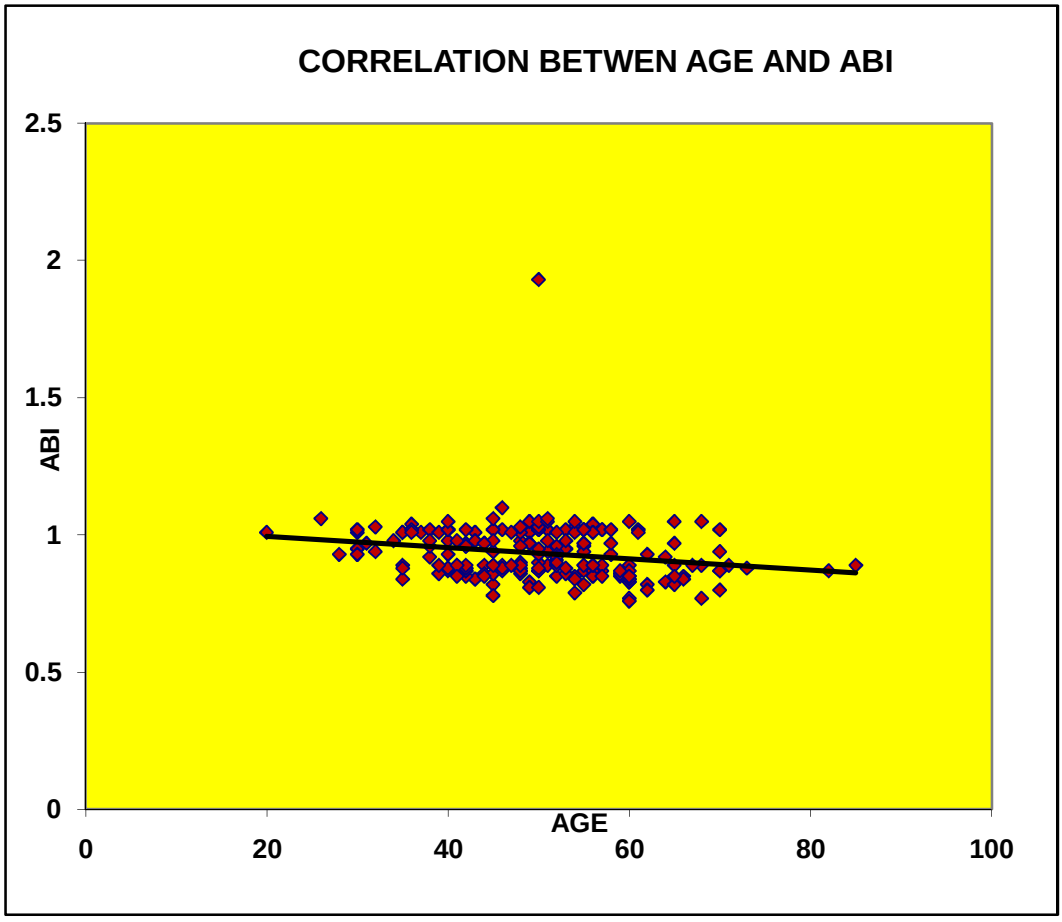


Fig 11:Correlation between age and ABI

The above scattered diagram shows the relation of ABI with the age of subjects. The correlation trendline indicates that there is decline of ABI with increase in age and this change was found to be statistically significant with p value of 0.003.

Comparison between age and ABI

Age	<=0.9	>0.9
21 - 30	0	100
31 - 40	27.6	72.4
41 - 50	50	50
51 - 60	50.7	49.3
61 - 70	60.9	39.1
71 - 80	100	0
81 - 90	100	0

Table 8: Comparison between age and sex of study subjects

Patients were sorted according to their age as shown in the above table. Percentage of patients with ABI <= 0.9 and those with >0.9 are determined in each group and plotted in the form of bar diagram.

Comparison between Age and ABI

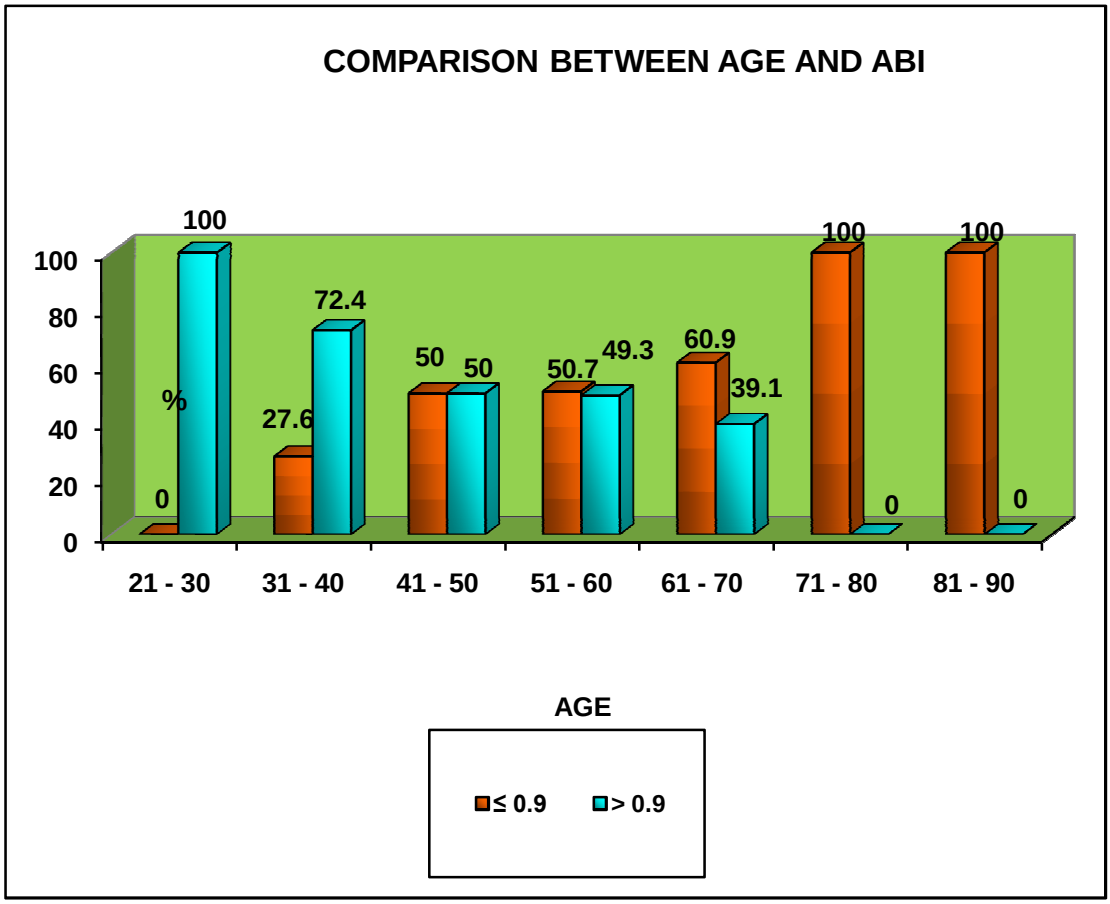


Fig 12: Showing comparison between age and ABI

Above diagram indicates that the percentage of patients with ABI <=0.9 increases as age increases.

Correlation between BMI and ABI

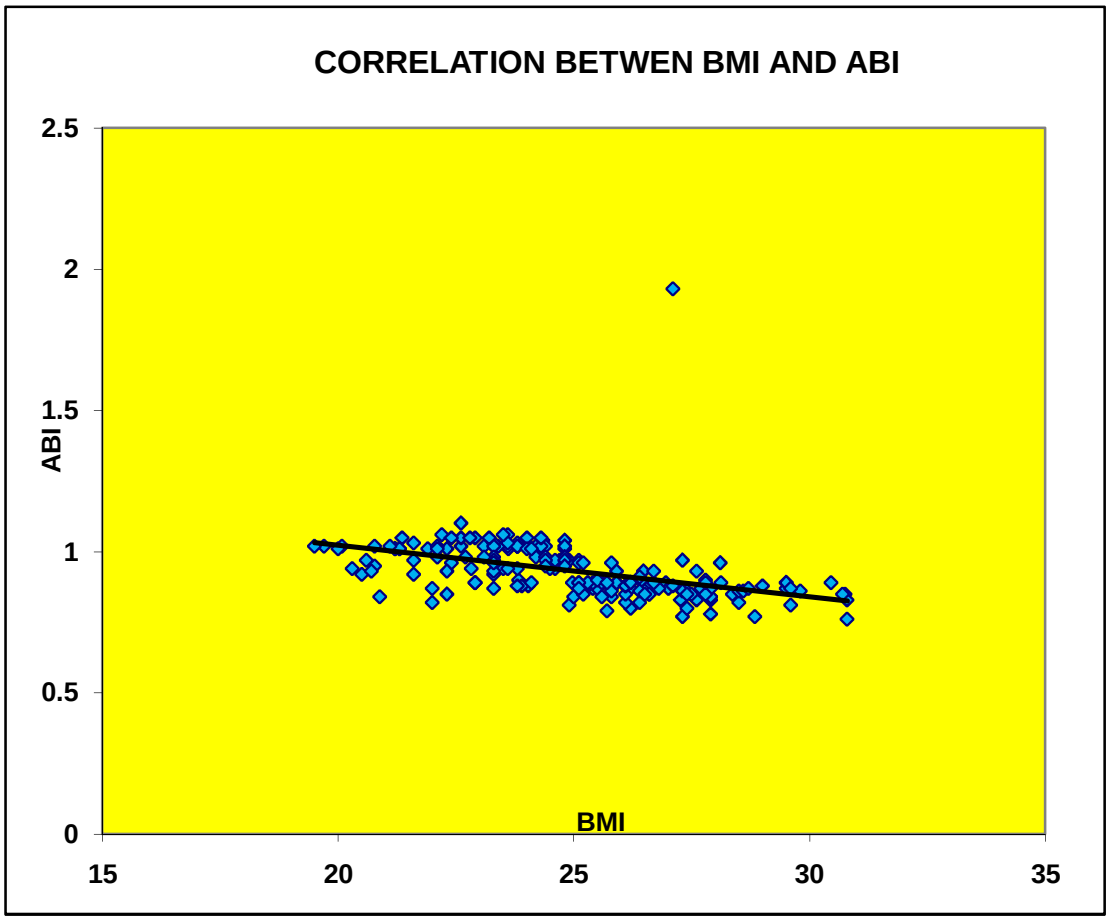


Fig 13: Correlation between BMI and ABI

This diagram shows us the correlation of ABI with BMI. The correlation trendline indicates that the ABI decreases with increase in BMI. This change is found to be statistically significant with p value of 0.0001.

Comparison between BMI and ABI

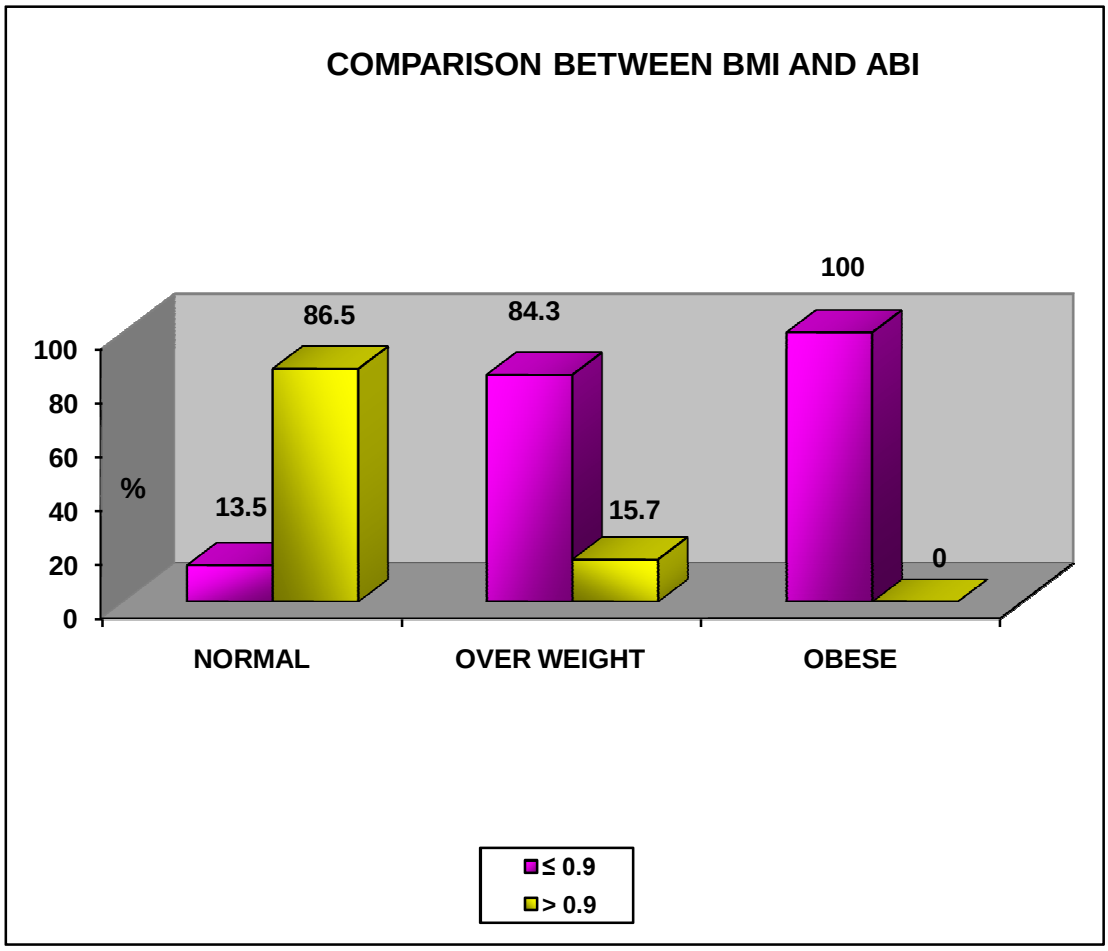


Fig 14 Bar diagram showing comparison between BMI and ABI

The above figure shows that percentage of patients with ABI ≤ 0.9 increases as BMI increases.

Correlation between hypertension duration and ABI

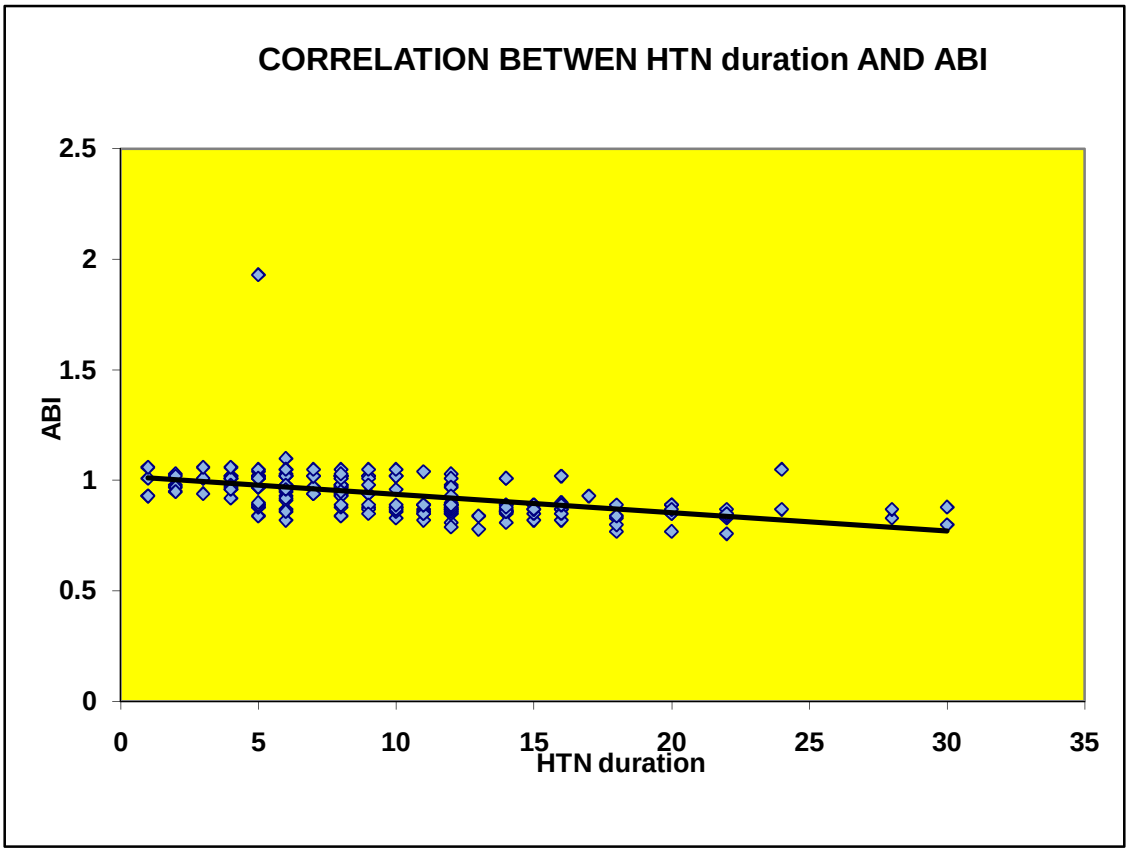


Fig 15:Correlation between hypertension duration and ABI

This scattered diagram shows the correlation between the duration of hypertension with ABI. The correlation trendline indicates that ABI decreases as duration of hypertension increases. This was found to be statistically significant with p value of 0.0001.

Comparison between hypertension duration and ABI

Duration	≤0.9	>0.9
1 - 5 YEARS	11.9	88.1
6 - 10 YEARS	23.2	76.8
11 - 15 YEARS	80.8	19.2
16 - 20 YEARS	86.4	13.6
21 - 25 YEARS	88.9	11.1
26 - 30 YEARS	100	0

Table 9: Comparison between hypertension duration and ABI

Percentage of patients with ABI ≤ 0.9 were calculated for various duration of hypertension as shown in the table. This is depicted in the bar diagram.

Comparison between hypertension duration and ABI

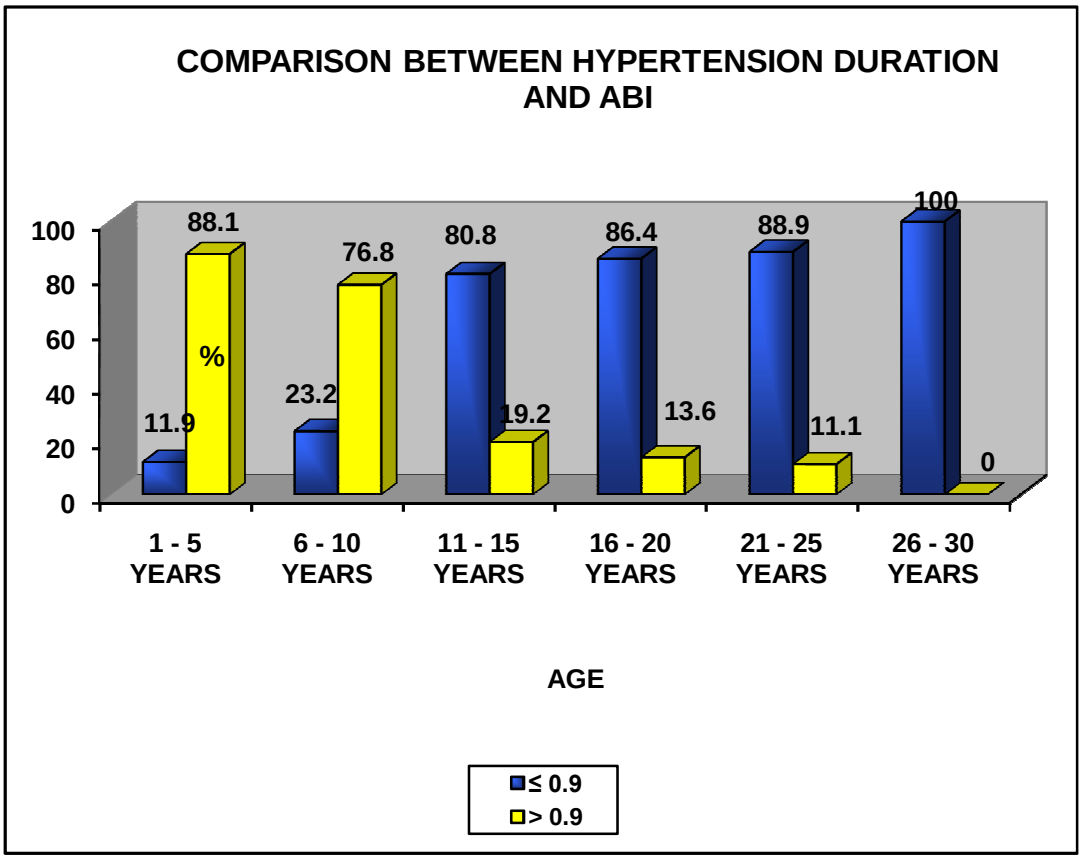


Fig 16: Comparison between hypertension duration and ABI

This diagram shows that percentage of patients with ABI≤ 0.9 increases as duration of hypertension increases.

Correlation between systolic blood pressure (SBP) and ABI

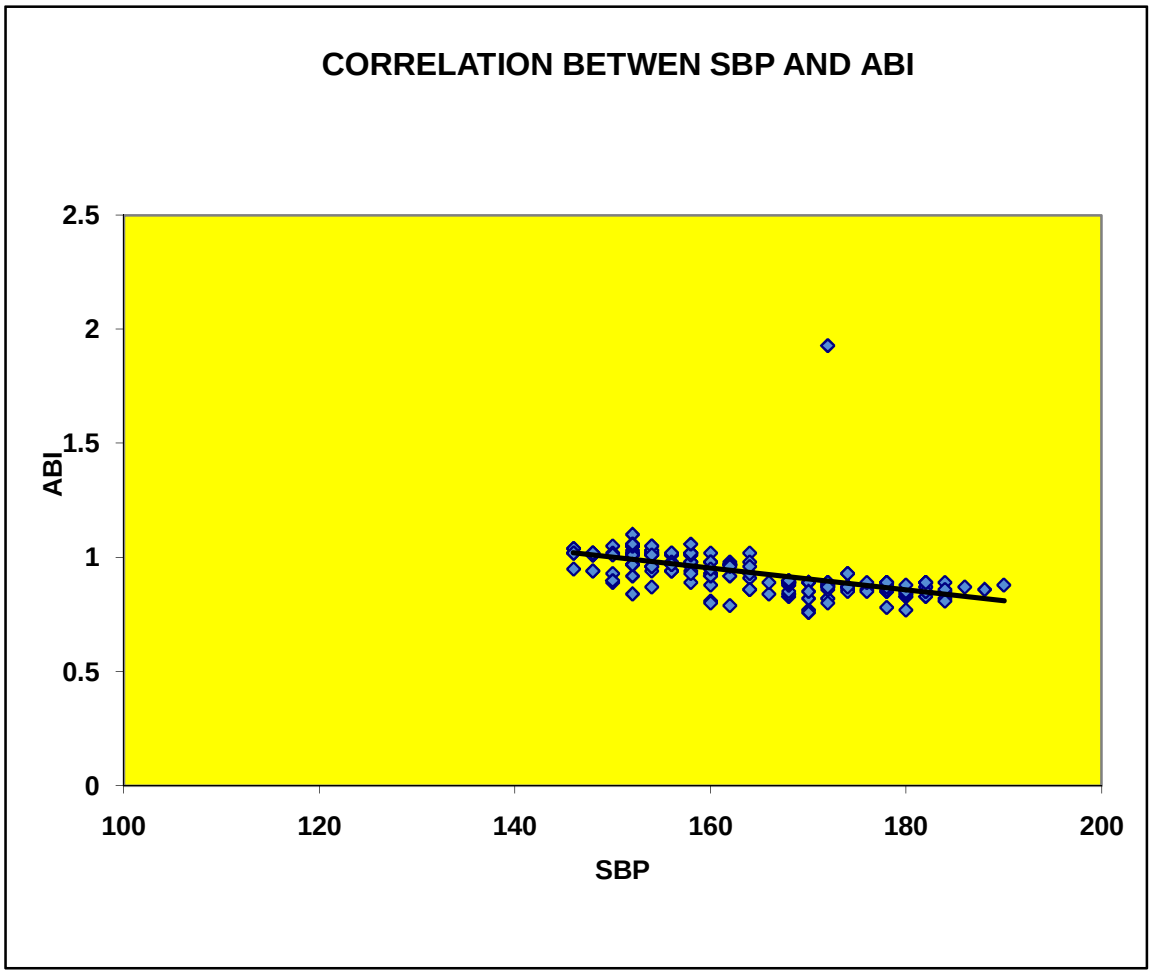


Fig 17: Correlation between systolic blood pressure and ABI

The above diagram shows that our patients fall between SBP of 146 to 190 mm Hg. It also depicts that as SBP increases ABI decreases which is statistically significant with p value of 0.0001

Correlation between diastolic blood pressure and ABI

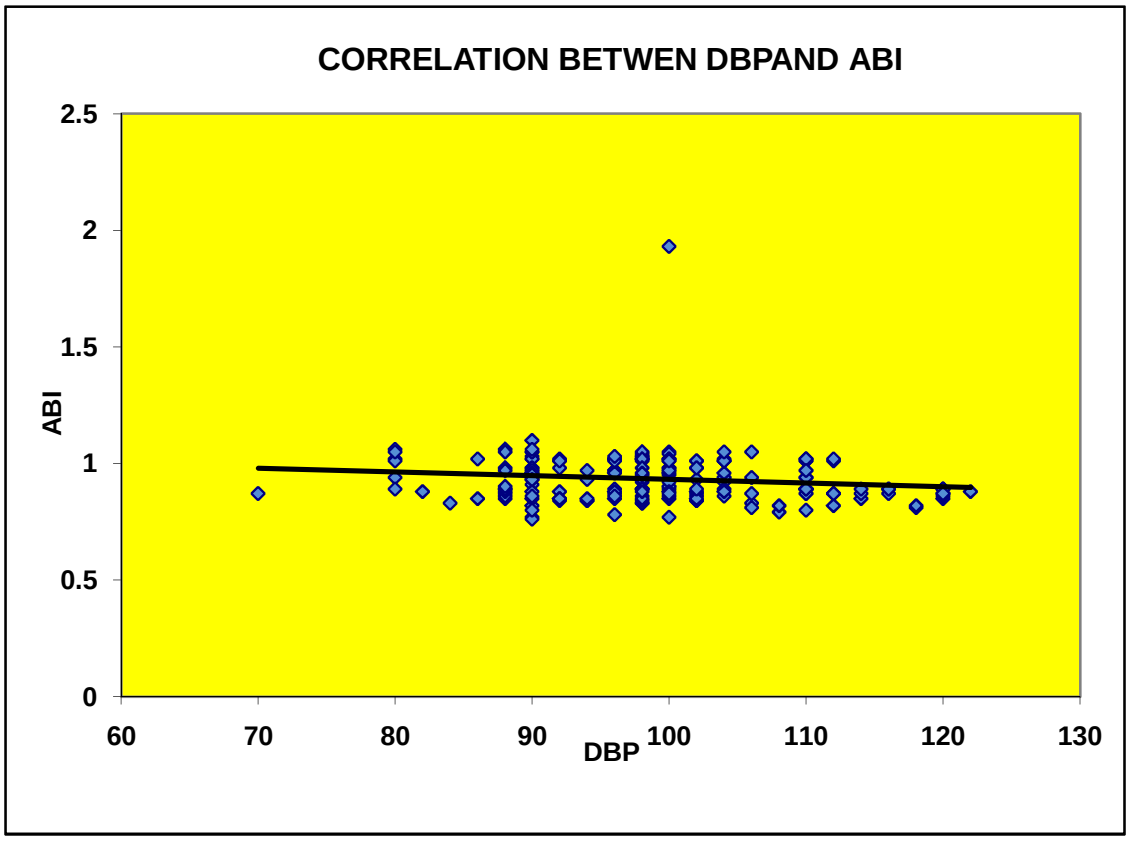


Fig 18 :Correlation between diastolic blood pressure and ABI

The above scattered diagram shows as DBP increases ABI decreases and this was found to be statistically significant with the p value of 0.039.

Correlation between pulse pressure (PP) and ABI

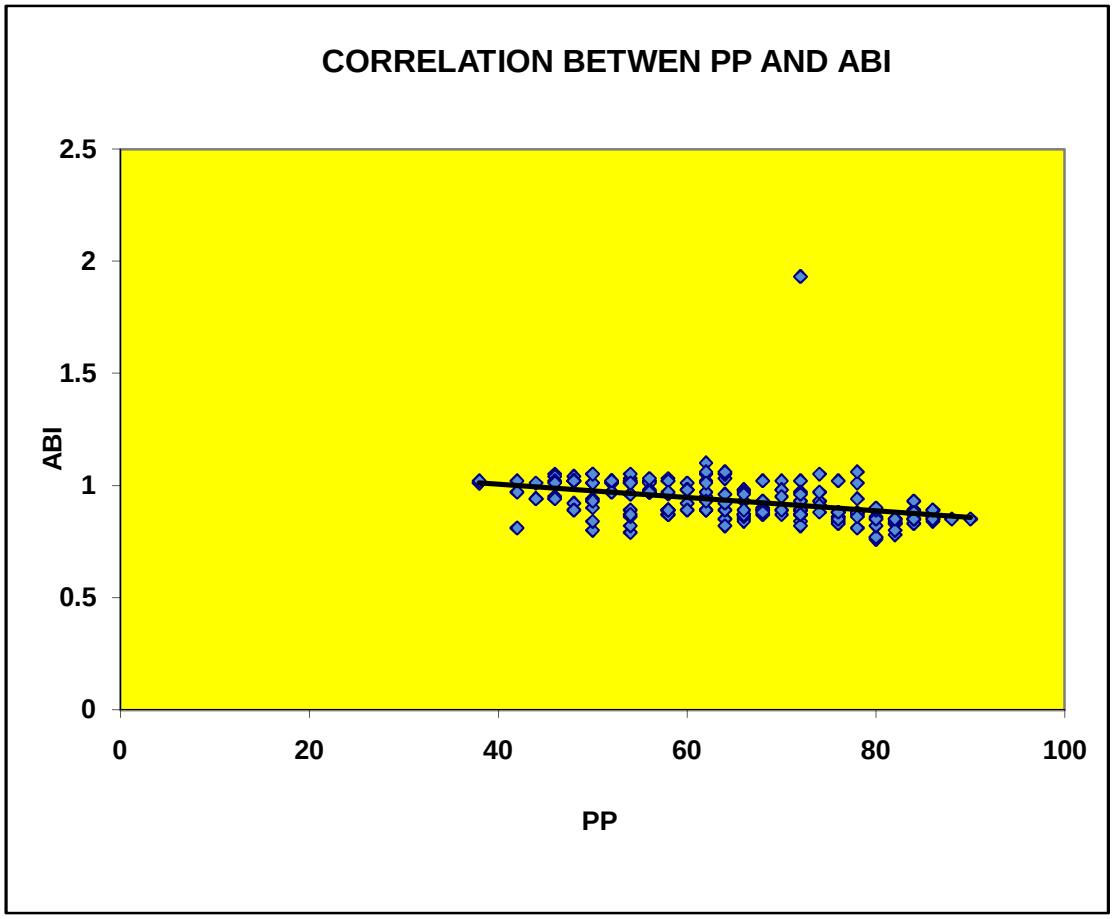


Fig 19 : Correlation between pulse pressure and ABI

This scattered diagram shows correlation between pulse pressure and ABI. The correlation gtrendline indicates that as pulse pressure increases ABI decreases. This was found to be statistically significant with the p value of 0.0001.

Comparison between sex and ABI

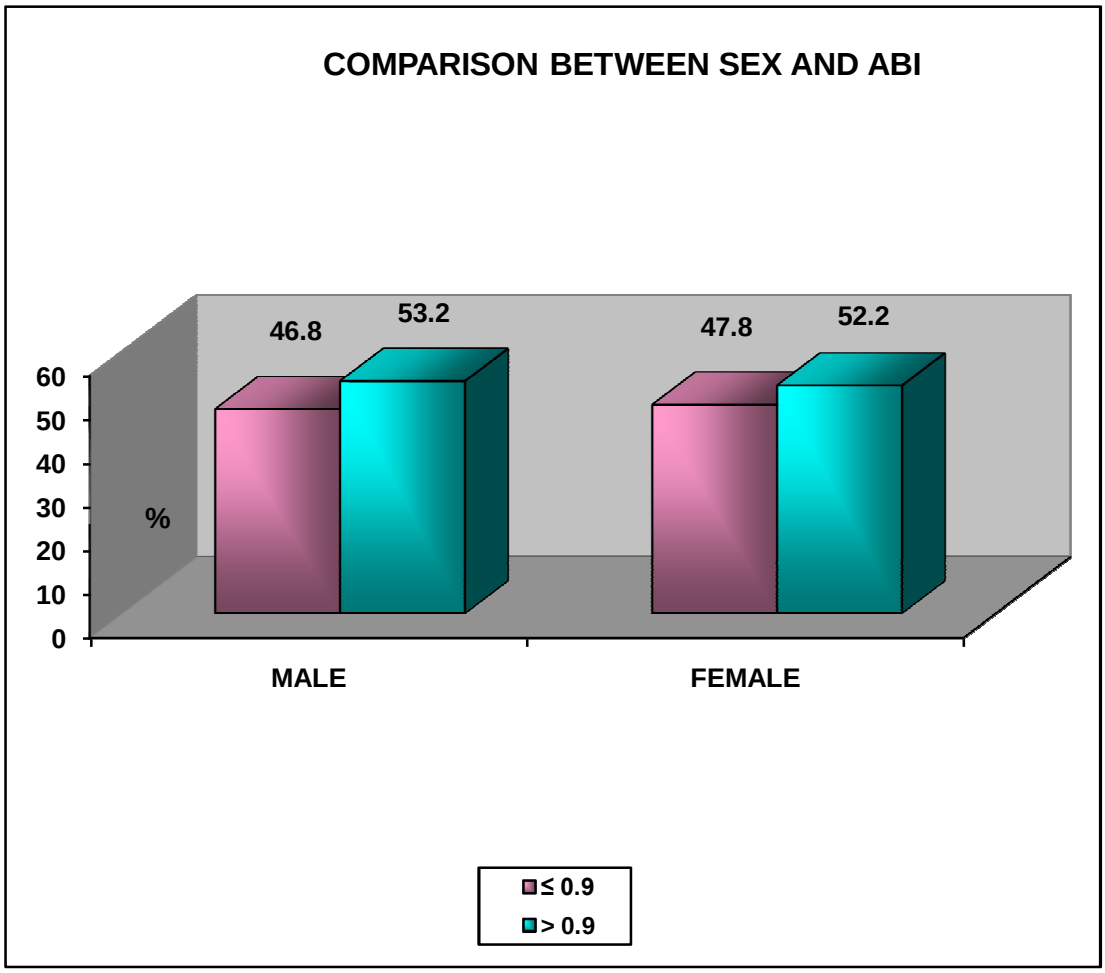


Fig 20: Comparison between sex and ABI

In this study 46.8% of males have ABI ≤ 0.9 and 47.8% of females have ABI≤ 0.9.


RESULTS

There were a total of 198 patients enrolled in this study.

- Prevalence of asymptomatic PAD was found to be 47%
- ABI was ≤ 0.9 in 46.8% males
- ABI was ≤ 0.9 in 47.8% females
- There was inverse correlation between age and ABI
- There was inverse correlation between ABI and BMI
- There was inverse Correlation between ABI and systolic pressure
- There was inverse correlation between ABI and diastolic pressure
- There was inverse correlation between ABI and pulse pressure
- There was inverse correlation between ABI and duration of hypertension.

CONCLUSION

CONCLUSION

Subclinical peripheral artery disease is common in hypertensive patients even though they do not have other co morbidities. Measuring the Ankle Brachial Index is an efficient method to detect patients who are at increased cardiovascular risk. It would help treating physician to advocate preventive measures to reduce the risk.

It would be beneficial to include Ankle Brachal index as a routine in primary care set up as it is safe, easy and patient friendly. Measuring ABI may significantly reduce the future cardiovascular events in hypertensive patients .

ANNEXURES

Abbreviations

ABI - Ankle Brachial Index

PAD – Peripheral Artery Disease.

CAD – Coronary Artery Disease.

CVD - Cardio Vascular Disease

IHD - Ischemic Heart Disease

ESRD – End Stage Renal Disease

BP – Blood Pressure

SBP – Systolic Blood Pressure

DBP – Diastolic Blood Pressure

PP – Pulse Pressure

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MASTER CHART

serial no.	name	age	sex	op no.	BMI	HTN duration	SBP	DBP	PP	ABI
1	Maya	40	F	2089	29.51	6	154	70	84	0.87
2	Kumar	56	M	2094	28.12	14	170	98	72	0.89
3	Amul raj	40	M	2073	27.6	6	174	102	74	0.93
4	Rukmani	58	F	2093	26.47	17	150	100	72	0.93
5	Astharalli	54	M	2091	30.75	14	184	120	64	0.85
6	Saraswathi	53	F	2095	20.77	6	146	100	46	0.95
7	Sundaram	60	M	2090	24.97	20	168	110	58	0.89
8	Jesi	60	F	2070	20.88	22	168	92	76	0.84
9	Ibrahim	44	M	2035	26.95	12	170	100	70	0.89
10	Divasigmani	73	F	2069	24.03	30	160	82	78	0.88
11	Kamatchi	51	F	2071	26.48	15	150	96	54	0.89
12	Prabha	36	F	2068	20.08	5	152	98	42	1.02
13	Lalitha	35	F	2072	23.61	5	148	110	38	1.01
14	Shanmuga sundaram	52	M	2067	28.37	12	180	114	66	0.85
15	Jothika	64	F	2088	30.8	28	180	98	82	0.83
16	K. kantha	45	F	2080	22.82	7	148	104	44	0.94
17	Abeya	68	F	2086	21.36	24	150	100	46	1.05
18	Janaki	48	F	2078	23.83	16	150	100	50	1
19	Sagunam	51	F	2084	20.77	10	146	100	54	1.02
20	Yousuf	55	M	2077	30.46	12	168	110	58	0.89
21	Padma	70	F	2083	21.99	28	174	96	78	0.87
22	Theresa	49	F	2076	27.26	10	180	98	82	0.83
23	Vasantha	36	F	2082	24.34	5	146	100	46	1.04
24	Vanaja	60	F	2075	28.84	20	170	90	80	0.77
25	Radha krishna	43	M	2081	25.8	8	166	94	72	0.84
26	Devaki	57	F	2087	23.8	16	150	96	56	1.02
27	Devdas	61	M	2074	23.6	8	158	90	58	1.02
28	Tamilselvi	44	F	2033	20.6	8	152	96	56	0.97
29	Shanmugam	54	M	2066	21.6	12	154	90	58	1.03
30	Kasthuri	55	F	2034	22.4	10	154	100	54	0.96
31	Nagarani	60	F	2045	30.8	22	170	90	80	0.76
32	Albert	57	M	2037	25.8	16	174	88	86	0.87
33	Renuka Devi	49	F	1952	24.9	12	160	118	42	0.81
34	Mohankumar	53	M	2038	23.4	8	160	90	48	1.02
35	Sangeetha	56	F	1886	24.8	11	146	98	48	1.04
36	Ranjitha	60	F	2039	27.9	18	168	84	84	0.83
37	Harilal	43	M	1887	22.1	3	148	110	38	1.01
38	Babu	52	M	2040	26.4	6	164	90	74	0.91
39	Pushpa	68	F	1889	27.3	18	180	100	80	0.77
40	Senthil raman	50	M	2044	27.8	12	168	100	68	1
41	Malliga	52	F	1875	24.1	14	150	100	44	1.01

42	Jayalakshmi	52	F	1880	23.8	9	156	110	78	0.94
43	Violet Mary	70	F	2041	26.2	30	160	110	50	0.8
44	Marhamma	50	F	1890	25.9	8	160	94	66	0.93
45	Sarasu	55	F	1884	24.3	9	148	96	76	1.02
46	Narasamma	38	F	2042	21.6	6	152	104	48	0.92
47	Ganesh moorthy	55	M	1951	27	12	174	102	72	0.87
48	vanalakshmi	54	F	1918	25.7	12	162	108	54	0.79
49	Mumtaj	61	F	1954	24.8	14	152	98	52	1.01
50	Rani	35	F	1915	24.1	4	156	112	50	1.01
51	Muthammal	57	F	1958	22.6	9	154	96	48	1.02
52	Andal	65	F	1894	26.1	16	172	108	64	0.82
53	Ranjini	54	F	2041	27.9	13	152	102	50	0.84
54	Mark	68	M	1960	25.6	18	168	104	64	0.89
55	Karthikeyan	45	M	2043	26.4	11	172	118	54	0.82
56	Jagadhammal	64	F	1873	23.3	12	162	98	60	0.92
57	Ramalakshmi	62	F	1920	22	6	170	90	80	0.82
58	Egavalli	42	F	1876	24.9	7	152	110	56	0.97
59	Chinnamma	55	F	1871	24.6	8	148	104	46	0.94
60	Kannan	51	M	1881	23.5	7	158	80	44	0.94
61	Prashanth	46	M	1882	22.6	6	152	90	62	1.1
62	Prema	37	F	1828	22.3	3	152	92	78	1.01
63	Anjalamma	36	F	1830	23.3	4	148	100	68	1.02
64	Rani	60	F	1824	27.6	22	182	106	76	0.83
65	Geetha	56	F	1825	26.7	16	172	114	58	0.87
66	Amaravathi	48	F	1827	25.8	12	174	120	54	0.86
67	Vaniyamma	49	F	1813	20	9	148	102	54	1.01
68	Devanesan	50	M	1816	19.7	5	156	80	72	1.02
69	Meenakshi	55	F	1819	24.4	7	146	98	70	1.02
70	Saroja	50	F	1787	24.8	8	146	104	46	1.02
71	Maliga	42	F	1789	26.6	11	178	90	88	0.85
72	Subalatha	20	F	1791	21.9	1	154	104	60	1.01
73	Sudha	32	F	1793	23.8	2	152	98	64	1.03
74	Elizabeth	49	F	1774	24	9	152	106	50	1.05
75	Elumalai	26	M	1776	22.2	1	152	88	78	1.06
76	Bharathi	48	F	1779	25.4	15	174	116	58	0.87
77	Meera	58	F	1782	24.7	8	152	96	72	0.97
78	Sakunthala	70	F	1785	20.3	9	156	110	46	0.94
79	Baby	58	F	1770	23.1	7	154	98	38	1.02
80	Sundar	45	M	1772	27.9	13	178	96	82	0.78
81	Mahalakshmi	35	F	1752	27.9	5	168	102	66	0.84
82	Murugesan	52	M	1755	25.8	8	158	104	54	0.96
83	Masilamani	50	M	1758	23.3	6	154	98	54	1.03
84	Sambulingam	82	M	1761	23.3	24	182	112	70	0.87
85	Kalyani	40	F	1763	22.7	6	160	100	66	0.98
86	Deepalakshmi	34	F	1694	24.8	2	158	92	66	0.98

87	Sumathi	38	F	1568	23.8	4	148	110	54	1.02
88	Nagalakshmi	42	F	1499	25.5	11	178	112	66	0.87
89	Valli	55	F	1765	28.5	15	184	112	72	0.82
90	Subramani	60	M	1798	25	22	180	98	82	0.84
91	Manaravi	39	M	1750	21.2	4	154	102	46	1.01
92	Arthi	32	F	1737	23.6	3	156	106	50	0.94
93	Subburayan	60	M	1748	22.6	6	152	88	62	1.05
94	Devi	36	F	1735	21.2	5	156	104	50	1.01
95	Mumtaj begam	45	F	1742	28.5	11	164	98	66	0.86
96	Ramani	45	F	1739	24.5	8	154	110	44	0.94
97	Varadhamma	66	F	1745	22.3	20	168	86	82	0.85
98	Elumalai samy	60	M	1943	26.2	22	178	112	66	0.87
99	Chellammal	62	F	1741	27.4	18	172	90	82	0.8
100	Karpagam	52	F	1717	23.3	8	160	98	50	0.93
101	Esther	50	F	1746	29.6	14	184	106	78	0.81
102	Jamuna	40	F	1722	24.2	6	152	96	56	1.02
103	Parimala	60	F	1708	30.7	22	182	100	82	0.85
104	Bharathiyammal	56	F	1710	21.3	8	158	104	52	1.01
105	Dhanaraj	40	M	1720	25.3	9	168	92	76	0.88
106	Rahimbee	40	F	1716	23.8	6	158	110	52	1.02
107	Krishnaveni	50	F	1688	22.4	7	152	98	54	1.05
108	Immali	59	M	1715	25.2	20	178	88	90	0.85
109	Lakshmi	46	F	1693	25.3	12	172	114	58	0.89
110	Mariya	50	F	1671	26.8	14	178	120	58	0.87
111	Mary	46	F	1673	24.3	8	158	110	58	1.02
112	Annamalai	71	M	1676	22.9	16	184	116	68	0.89
113	Nagamma	39	F	1670	27.4	6	188	102	86	0.86
114	Ettiyappan	30	M	1677	21.3	4	152	96	56	1.01
115	Jalandhara	51	F	1663	24.4	8	158	102	60	0.98
116	Govindhammal	65	F	1664	25.4	16	178	88	86	0.89
117	Vijayakumar	30	M	1668	23.8	2	164	112	48	1.02
118	Irudhayam	45	M	1650	23.3	8	156	98	48	1.02
119	Radha	42	F	1669	27.7	9	172	106	66	0.87
120	Renuka Devi	53	F	1651	24.2	12	162	102	56	0.98
121	Ayyanar	51	M	1653	22.9	8	152	90	62	1.05
122	Nagarajan	66	M	1660	25.6	18	180	94	86	0.84
123	Jayaraman	57	M	1632	22.9	11	168	98	70	0.89
124	Thilagavathi	48	F	1633	24.4	8	164	98	60	0.98
125	Mani	45	M	1636	23.3	9	160	100	60	0.98
126	Gowri	55	F	1638	25.9	14	178	110	68	0.89
127	Rosy	43	F	1639	24.8	8	158	88	66	0.98
128	Gibisha	45	F	1644	25.1	11	182	110	72	0.89
129	Boopathy	54	M	1612	24	12	158	80	54	1.01
130	Rooja	44	F	1645	27.3	14	184	104	80	0.86
131	Rajammal	65	F	1615	24.6	12	162	90	52	0.97

132	Vadivel	41	M	1646	25.3	9	182	98	84	0.89
133	Padhamma	35	F	1617	29.5	5	178	120	58	0.89
134	Chandra	42	F	1620	23.9	10	168	90	78	0.88
135	Shanthi	28	F	1599	22.3	1	164	90	84	0.93
136	Pencillamma	70	F	1600	19.5	10	148	100	48	1.02
137	Raghu	42	M	1602	21.1	6	154	92	52	1.02
138	Sakunthalammal	48	F	1587	26.6	10	178	102	76	0.88
139	Selvi	45	F	1591	26.5	10	176	90	86	0.86
140	Valliyammal	41	F	1595	22.1	4	156	90	70	0.98
141	Sajin	31	M	1588	24.8	2	156	100	56	0.97
142	Munniyammal	59	F	1540	29.8	14	178	100	78	0.86
143	Vevekannadha	35	M	1528	26.1	5	172	96	76	0.88
144	Raju	44	M	1543	27.5	11	180	96	84	0.85
145	Jyothilakshmi	30	F	1532	24.8	2	160	98	62	0.95
146	Sarojini	56	F	1530	26.6	16	180	94	86	0.85
147	Raja subramani	57	M	1536	26.1	12	170	92	86	0.85
148	Mythili	38	F	1401	28.1	6	154	96	58	0.96
149	Jaya	59	F	1435	26.4	14	164	88	76	0.86
150	Violet	55	F	1418	22.1	5	158	86	62	1.02
151	Ranjana	49	F	1510	23.3	5	162	88	66	0.97
152	Anjamma	45	F	1511	23.6	3	158	80	62	1.06
153	Mary joseph	49	F	1485	21.6	6	162	100	62	0.97
154	Kasthuriyammal	40	F	1512	26.1	5	172	88	84	0.88
155	Kumar babu	42	M	1489	23.8	8	180	104	76	0.88
156	Alamelu	48	F	1513	24.1	8	154	92	62	1.01
157	Neelaveni	38	F	1498	23.1	6	160	90	70	0.98
158	Shiva	52	M	1514	20.5	4	160	100	64	0.92
159	Kalavathi	54	F	1521	24.3	10	154	104	64	1.05
160	Saroja	48	F	1469	27.7	10	186	120	66	0.87
161	Kasthuri rani	50	F	1452	27.1	5	172	100	72	1.93
162	Pooniyammal	46	F	1471	27.9	12	182	110	72	0.87
163	Mary karvallo	42	F	1464	25.9	11	182	110	72	0.89
164	Satidevi	41	F	1478	26.7	10	172	110	62	0.89
165	Karpaga veni	55	F	1447	27.3	5	152	94	74	0.97
166	Fathima	60	F	1450	27.8	15	182	102	80	0.85
167	Pappa	53	F	1453	28.6	12	172	96	76	0.86
168	Gunasheeli	41	F	1460	27.4	9	174	98	76	0.85
169	Poongodi	46	F	1446	27.1	12	172	98	74	0.88
170	Shamila	30	F	1430	26.7	1	174	90	68	0.93
171	Ramabai	48	F	1445	29.5	16	168	110	58	0.89
172	Nirmala	44	F	1436	25.1	4	162	110	42	0.97
173	Devika	50	F	1427	28.7	12	178	110	68	0.87
174	Baskaran	65	M	1423	23.2	5	152	90	50	1.05
175	Sakunthaladevi	50	F	1433	26.1	12	176	100	76	0.88
176	Natarajan	45	M	1428	27.8	8	178	120	58	0.89

177	Malini	42	F	1432	25.1	4	164	98	72	0.96
178	John	48	M	1425	23.6	8	154	96	56	1.03
179	Rathinammal	42	F	1441	25.2	4	162	90	64	0.96
180	Mary jawahar	85	F	1437	25.1	20	158	110	48	0.89
181	Rajeshwari	52	F	1421	26.2	11	168	102	66	0.89
182	Samanthi	39	F	1403	26.2	5	182	120	62	0.89
183	Amudha	40	F	1406	22.8	6	154	80	74	1.05
184	Ellappa	67	M	1381	22.9	15	166	80	86	0.89
185	Paul raj	52	M	1384	24.1	20	172	110	62	0.89
186	Arjunan	55	M	1388	24.4	5	156	90	58	0.97
187	Sekar	48	M	1354	23.3	6	154	90	66	0.96
188	Durai	47	M	1359	25.6	12	168	88	80	0.89
189	Rajan	50	M	1363	24.4	8	160	90	70	0.95
190	Ranganathan	62	M	1399	20.7	12	158	90	62	0.93
191	Pasupathi	47	M	1416	22.1	5	154	100	54	1.01
192	Kala	53	F	1521	29	14	190	122	68	0.88
193	Johnson	51	M	1497	23.5	4	152	90	64	1.06
194	Allis	70	F	1407	25.1	20	172	100	72	0.87
195	Annammal	65	F	1397	26.5	22	176	92	84	0.85
196	Shivaraj	52	M	1517	25.5	5	168	88	80	1
197	Jeyakumari	59	F	1414	29.6	15	174	120	54	0.87
198	Panchali	56	F	1455	25.7	12	176	116	60	0.89

INSTITUTIONAL ETHICAL COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Ref.No.1589/ME-1/Ethics/2014 Dt:06.03.2014.
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A Study of ankle brachial index in systemic hypertension" – For Project Work submitted by Dr.Deepa Avadhani, MD (GM), PG Student, KMC, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

29/5/14.

CHAIRMAN,
Ethical Committee
Govt.Kilpauk Medical College,Chennai

GOVT. KILPAUK MEDICAL COLLEGE
29 MAY 2014
Kilpauk, Chennai-10.